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LXXII

CARCINOMA OF THE LARYNX AND TOTAL LARYNGECTOMY*

S. J. CROWE, M.D.
AND
EDWIN N. BROYLES, M.D.

BALTIMORE, MD.

There are only two methods of treating a patient with carcinoma of the larynx. One is irradiation with roentgen-rays or radium. This is a valuable aid in the treatment of malignant growths, but should be used only in inoperable cases or to supplement operative removal. At the present time the results of ray therapy are so erratic and unpredictable, that whenever the diagnosis of cancer has been established from the history, examination and biopsy, and the growth is still localized and can be safely removed, the operation should not be postponed. The fact that the majority of carcinomas of the larynx are of the squamous cell, pearl forming variety which is very resistant to radium treatment adds force to this argument. We have all seen tumors of the tonsil or larynx permanently cured with irradiation therapy, but we have also seen tumors in the same location and

* From the Department of Otolaryngology of the Johns Hopkins University and Hospital.

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with the same histologic appearance that temporarily improved or even disappeared for a time, only to recur locally or in the adjacent lymph glands. If this irregular response is due to a variation in the dosage, the method of application or the screens used, then irradiation treatment is still in the experimental stage. There are many patients with inoperable conditions, and until the results of x-ray or radium treatment are more consistent and encouraging in these cases an operable new growth in the larynx should never be treated with irradiation alone.

The other method is surgical removal. The general principles of the operation are the removal of the growth with an adequate margin to insure against recurrence, and to preserve as far as possible the voice and the normal channels for breathing and swallowing. In carefully selected cases laryngo-fissure is the operation of choice. By laryngo-fissure we mean removal in one piece of the affected vocal cord, including its attachment to the arytenoid cartilage, the entire anterior commissure area and a large portion of the thyroid cartilage. It would perhaps be more accurate to describe this operation as excision of a growth in the larynx by the laryngo-fissure route. It differs from a hemilaryngectomy in that the posterior half of the cricoid is not removed. The perichondrium should be carefully preserved and, after the affected half of the larynx is removed, stitched over the exposed neck muscles to form a smooth lining for the new larynx, thus preventing granulations and increasing the chances for a good voice. Removal by the laryngo-fissure route is contraindicated if the growth involves the anterior commissure, has fixed the true cord, or has spread to the ventricle or false cord. In all borderline cases where there is any doubt about the extent of the growth the entire larynx should be removed.

As a result of our experience, we have evolved the following one-stage operation for total laryngectomy which is simple, free from shock and other postoperative complications. The success of the operation depends upon the most rigid adherence to the following rules:

1. The growth must be operable or intrinsic. The operation we propose is not suitable when the cartilages are invaded, the false cord is involved or the growth has spread to the muscles and glands of the neck.

2. The general physical condition must be such that the patient is a reasonably good operative risk.

3. Great emphasis is placed on preoperative elimination of infection in the sinuses, tonsils and particularly the teeth and gums.

4. The anesthesia is a combination of avertin, procain and chloroform. Avertin relieves the patient of much apprehension, and if not more than 75 mg. per kilo is given it in no way interferes with the postoperative cough reflex. Very little chloroform is necessary and its use prevents almost entirely bronchial irritation and excessive mucus secretion after the operation. If the growth interferes with breathing a tracheal tube is inserted under local anesthesia before avertin is given; if not, the trachea is opened at a later stage in the operation.

5. The operative procedure is based on sound surgical principles. It is as simple as possible. Every effort is made to avoid traumatization with clamps, retractors or sutures. Bleeding is controlled with ligatures, instead of the high frequency cautery, because wound infection and a prolonged, uncomfortable convalescence often follow the use of a cutting or coagulating current. A closure is made that prevents leakage of mouth secretions into the wound or trachea. We avoid pockets or dead spaces; we use the smallest size chromic catgut, and the only drain left in the wound is a piece of rubber tissue smaller than a pencil, flattened and placed in the midline. A little of this drain is pulled out and cut off each day and removed entirely on the third day after operation. The wound is not probed, suctioned or irrigated, because these procedures introduce skin organisms.

6. Before the mucous membrane flap closing off the pharynx is sutured in place, a small, soft rubber catheter is passed through the nose and into the esophagus. This catheter extends only half way down the esophagus. Fluids are given intravenously during the first 24 hours, but after this are introduced into the catheter with a syringe. On the third day after operation if there is no evidence of leakage or wound infection the feeding tube is removed and the patient given a soft diet.

During the operation the foot of the table is elevated about 18 inches and, in addition, suction is used to prevent aspiration of blood or mouth secretions. After the operation the patient is made as comfortable as possible in bed with two or more pillows under his head. A special nurse is required to keep a moist cloth over the tracheal tube and with suction to keep his trachea clear. During the postoperative period these patients need rest, and too much nursing and too many dressings are often detrimental.

The wound is tightly closed with the exception of one small drain in the midline, but it is carefully watched, and at the first sign of infection the skin sutures are removed and the superficial layers opened. This occurs with surprising infrequency.

The illustrations show the operative technique. If the operation is properly done there is no loss of blood, no shock and the patient usually sits up or gets out of bed on the second day. The feeding tube is removed on the third day and the patient is usually able to leave the hospital two weeks later. The incidence of postoperative complications, shock, pneumonia, cough and a profuse mucopurulent tracheal discharge are in direct proportion to the care with which the above rules are followed.

SUMMARY

The cases for operation must be carefully selected. When the growth has invaded the cartilage, muscles or glands we use irradiation therapy only. All intrinsic malignant growths are removed either by the laryngo-fissure route or by total laryngectomy. A laryngectomy is chosen when the anterior commissure is involved or the vocal cord is fixed. The end results of laryngo-fissure have been disappointing in our hands, due, we think, to improper selection of of the cases.[1]

Other important factors which insure a good result are: simple surgical principles as regards traumatism of tissues, hemostasis, and postoperative treatment; great care to close the pharynx tightly so as to prevent leakage of mouth secretions into the wound or trachea. Fluids are given through a small, soft rubber nasal catheter which extends only half way down the esophagus. This catheter is removed on the third day if there is no leakage of mouth secretions into the wound; the patient is encouraged to get out of bed on the second or third day; a special nurse is in constant attendance until the feeding tube is removed and longer if tracheal secretions are troublesome. The results of x-ray or radium treatment while sometimes very effective are still too uncertain to warrant their use in place of surgery when the growth is operable, but may be a valuable aid after operation. The dissection is made with a knife and hemostasis with the smallest chromic catgut ties rather than the high frequency cautery.

REFERENCE

1. Broyles, Edwin N.: Late Results Following Operation for Carcinoma of the Larynx. Archives of Otolaryngology, 24:475-483 (Oct.), 1936.

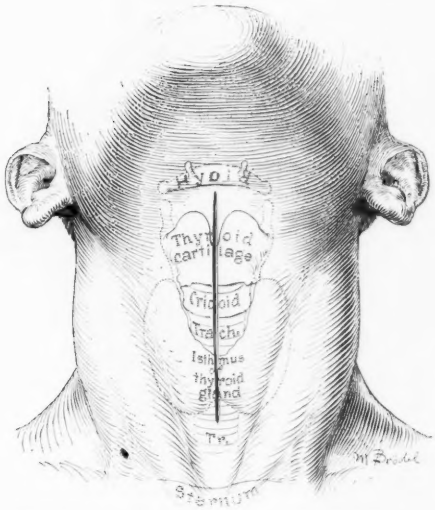


Fig. 1. The incision is in the midline and extends from the hyoid to the lower border of the thyroid isthmus.

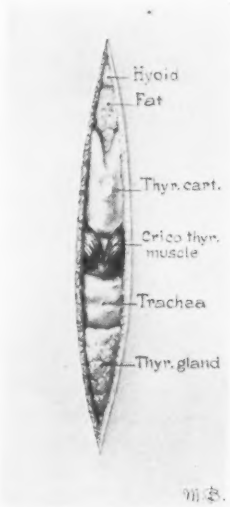


Fig. 2. The sterno-hyoid and sterno-thyroid muscles are gently pushed aside exposing the thyroid cartilage, crico-thyroid muscles, trachea and thyroid gland.

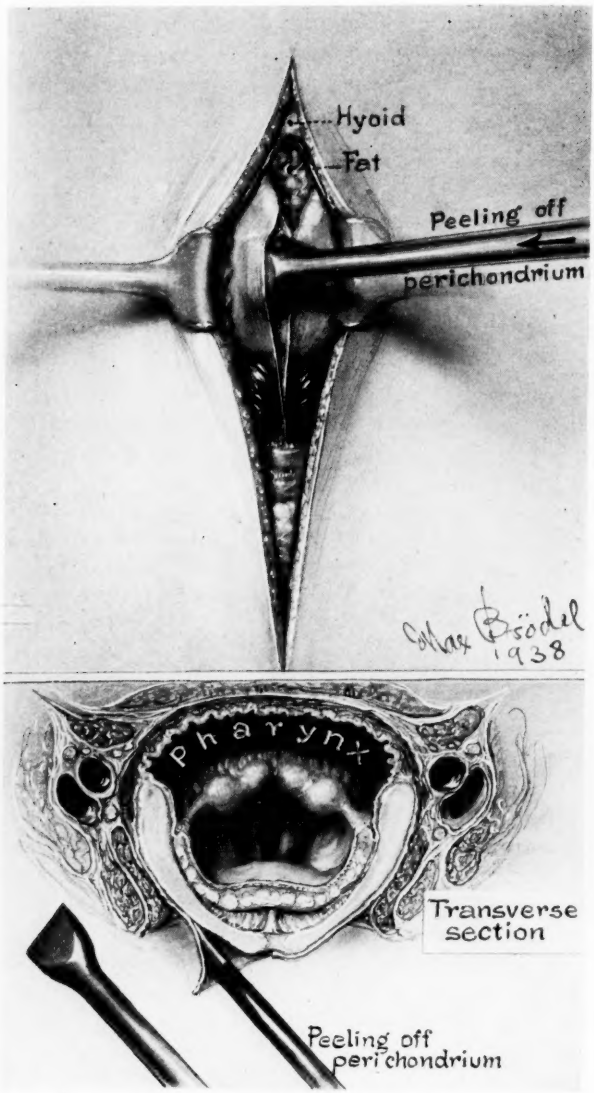


Fig. 3. To avoid crushing or tearing the neighboring muscles and fascia the larynx is skeletonized by peeling off the perichondrium, and everywhere making the dissection as close to the trachea as possible.

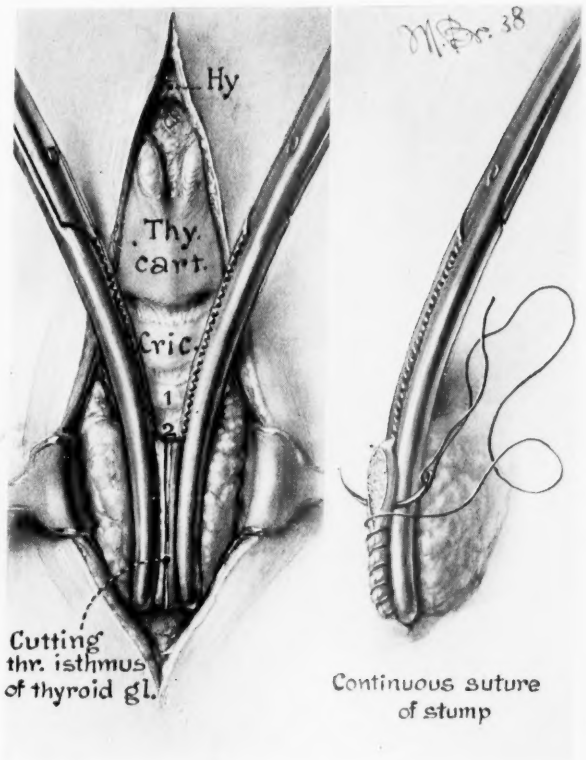


Fig. 4. Method of dividing and ligating the isthmus of the thyroid gland.

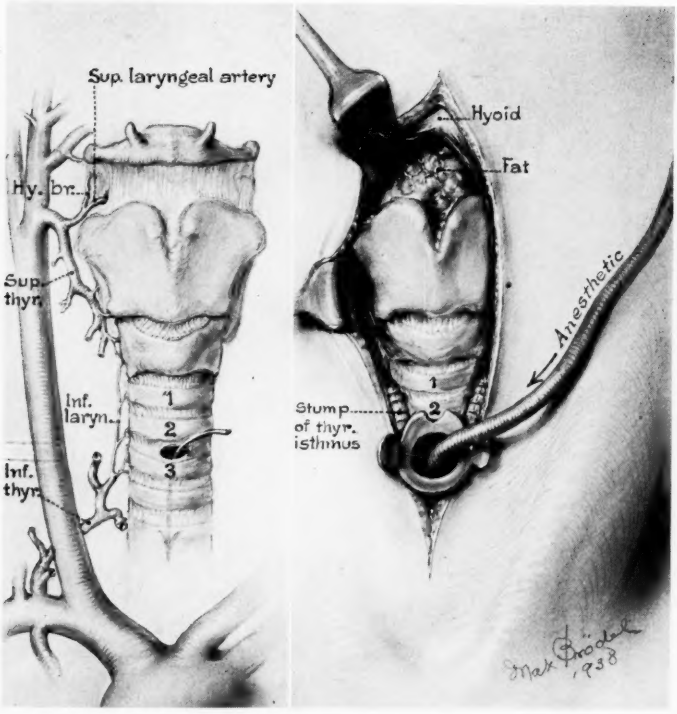


Fig. 5. The anatomical drawing on the left shows the relation of the superior thyroid artery to the superior laryngeal and other vessels supplying the larynx; also the level of the transverse incision for the tracheal tube. On the right is shown the tracheal tube; the small rubber tube through which chloroform vapor is blown; the thyroid isthmus which has been divided and sutured, the retractors placed for exposure of the cornu of the thyroid and ligation of the vessels lying deep in this angle.

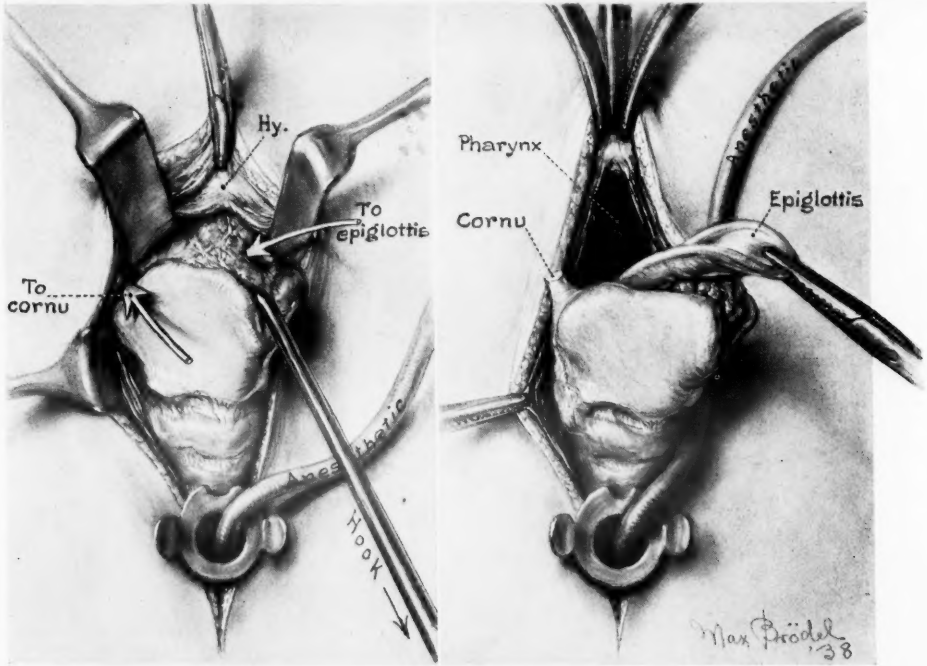


Fig. 6. By rotating the larynx, the cornu first on one side and then on the other is exposed and cut, the artery and vein ligated with chromic catgut (size 00), and the pharynx opened by dividing the thyro-hyoid membrane. The interior of the larynx is inspected for the subglottic extent of the growth, after which the pharynx and larynx are both packed with gauze to prevent aspiration of blood or mouth secretions.

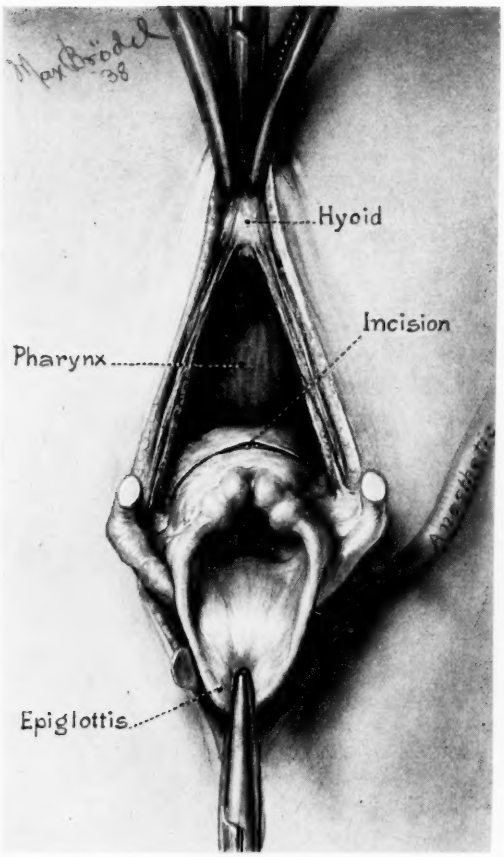


Fig. 7. The mucous membrane on the posterior surface of the larynx and in the pyriform sinuses is carefully removed and used to close the pharyngeal defect.

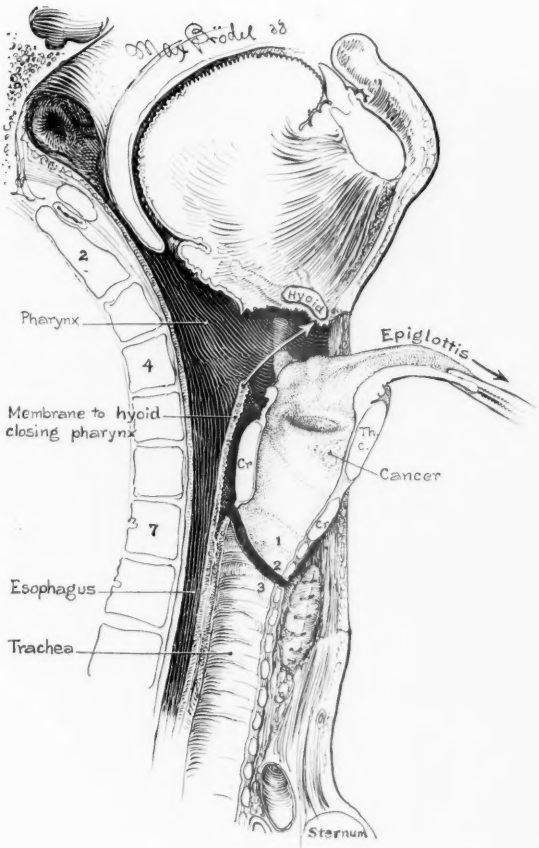


Fig. 8. To show how much is removed (the epiglottis, the entire larynx, a part of the cornu of the thyroid cartilage and a part of the first and second tracheal rings) and how much is left in place. In making this dissection every effort is made to avoid injury to surrounding tissues and the formation of pockets.

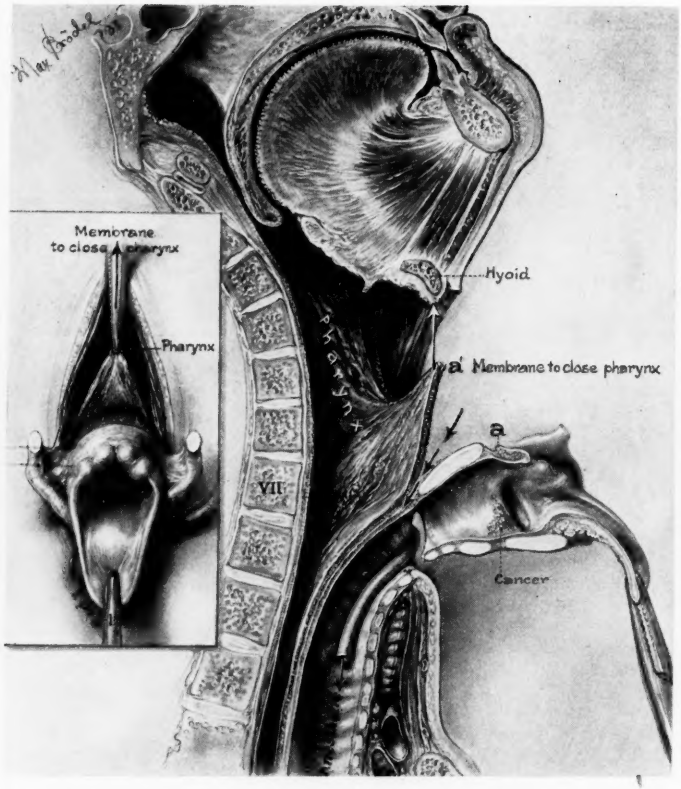


Fig. 9. Inset shows dissection of membrane and size of the pharyngeal defect to be closed. Just before the larynx is cut free the trachea is pulled forward and anchored to the surrounding muscles as shown in Fig. 10. Chromic catgut (size 0) is used.

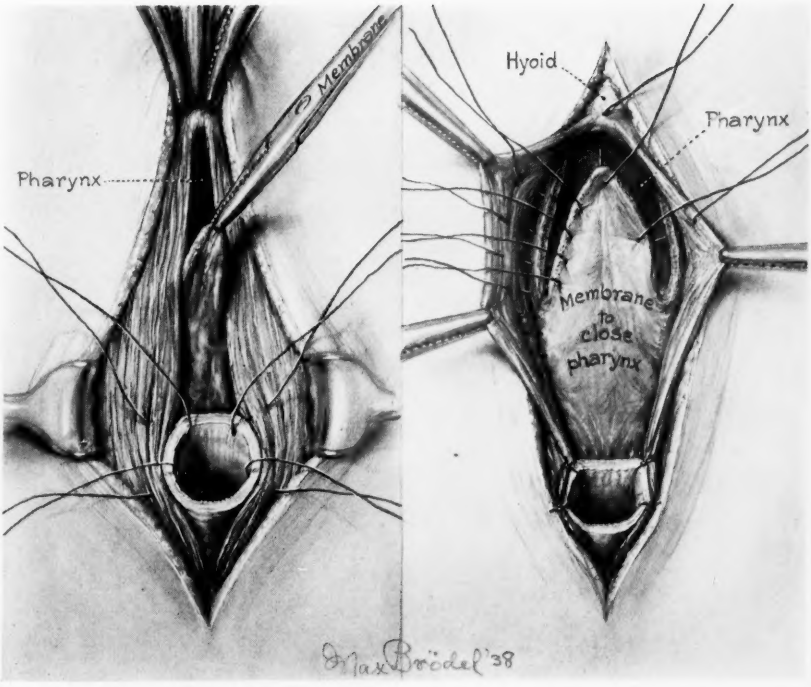


Fig. 10. The most important feature of the operation is shown on the right: interrupted chromic catgut sutures (size 00) include the mucous membrane flap, the mucous membrane of the pharynx and a little of the under surface of the sterno-hyoid muscle. When tied the muscle covers the line of incision and prevents leakage of saliva or food into the neck wound. To get this result, however, the wound must be closed without tension on any of the sutures.

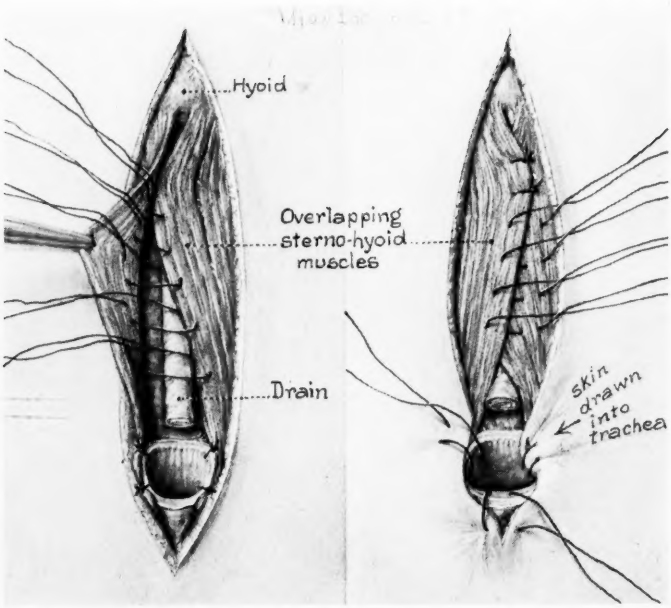


Fig. 11. One small, flattened roll of rubber tissue is placed in the midline. This is the only drain necessary, since no pockets or dead spaces have been left anywhere in the neck. The wound is then reinforced by overlapping the sterno-hyoid and the remains of the sterno-thyroid muscles. After removing all subcutaneous fat the skin is drawn into the trachea and loosely sutured. The tracheal tube, thickened with tape, as shown in Fig 12, holds this skin in place.

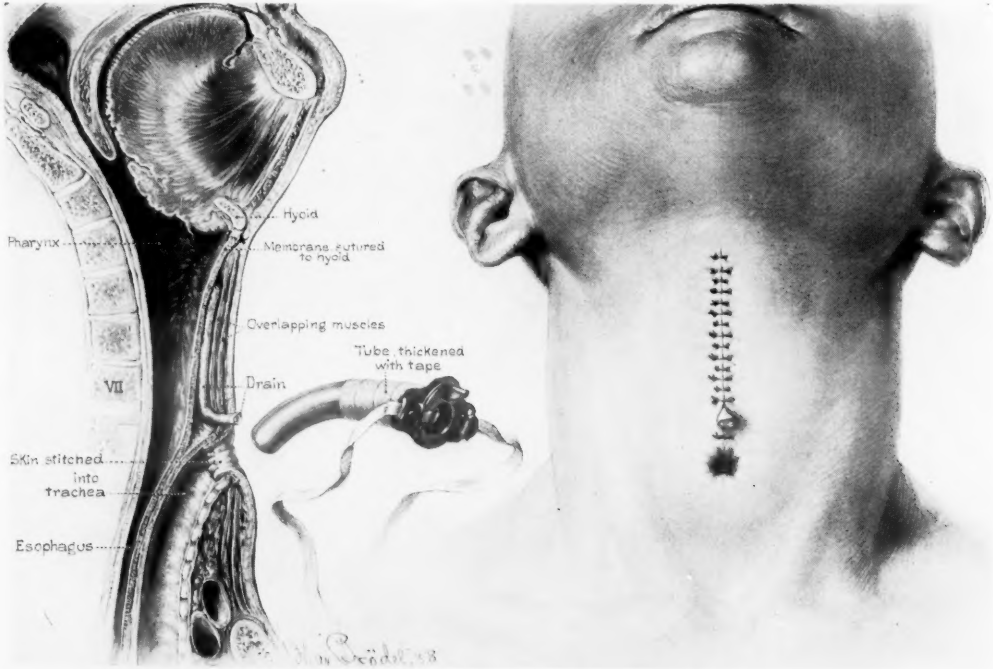


Fig. 12. The wound is closed but the skin incision must be opened at the first sign of infection. If there is no infection the rubber tissue drain is gradually withdrawn and removed entirely on the third day. If the tracheal opening ever becomes too small the skin around the lower half is elevated and the underlying anterior wall of the trachea removed with a high frequency cutting current.

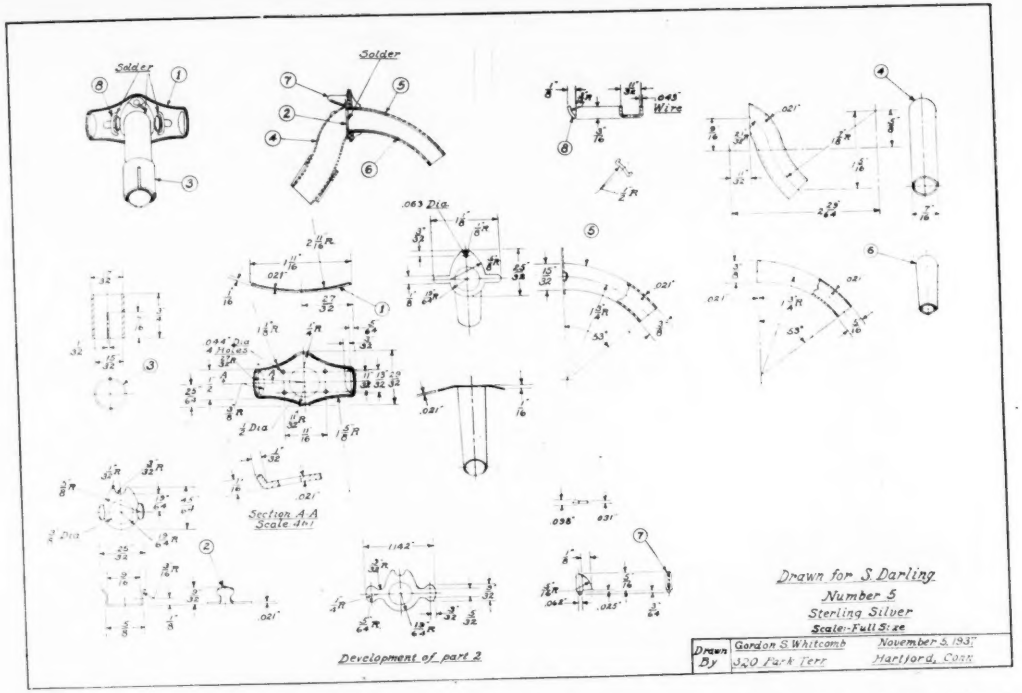


Fig. 13. Details for the construction of a tracheal tube to be used with an artificial larynx. It was designed by a patient who finds it very comfortable and convenient.

LXXIII

TREATMENT OF CHRONIC STENOSIS OF THE LARYNX WITH SPECIAL REFERENCE TO SKIN GRAFTING*

V. E. NEGUS, M.S., F.R.C.S.

LONDON

In submitting this paper to so distinguished an assembly of laryngologists, I have more than one object in view. I wish to give a brief account of the various measures adopted by some of my colleagues and myself in dealing with these difficult cases, with some indication as to which methods appear to be the best. I also hope that the subject will be clarified by subsequent discussion.

The variety of methods of treatment is an indication of the difficulties met with by us all. The type of case now under discussion must of course be limited to chronic conditions, in which any active disease, such as syphilis, has been arrested. It seems best to subdivide the subject according to methods of treatment and not to consider together various types of stenosis, as certain cases, similar in causation, require different remedies.

There are so many references to the subject in the literature that it has been found impracticable to refer in detail to them here; I must apologize for this omission.

Repeated Bouginage: I have tried this form of treatment in certain instances with various types of dilators, but without much success; any improvement has been of temporary duration only. The use of electrically heated bougies is said by Dean and Johnston to offer advantages, but I have no personal experience of the method.

Division of Webs With Cautery or Knife: If adhesions form over a wide area it is useless merely to divide them, unless means are taken to keep the raw surfaces apart until healing is complete.

Rare cases are seen where a thin web obstructs only the anterior part of the glottis and this can be divided easily. Great care, however, must be taken to avoid the production of fresh adhesions, particularly if diathermy is used. One or two difficult cases have been

*Read by invitation before the American Laryngological Association, at the meeting held in Atlantic City, May, 1938.

Fig. 1

Fig. 2

Figs. 1 and 2.—*Permanent Tracheostomy.*—The patient at the age of five was operated on for removal of tonsils. One week later she suffered from dyspnea with purulent bronchitis. Tracheotomy was performed, but subsequent attempts at removal of the cannula failed. Dilatation, followed by intubation, was tried. She was later referred to me with much swelling of the aryteno-epiglottic fold on the right side. The tracheotomy tube was replaced at a lower level. There is now fixation of both arytenoid cartilages and attempted removal of the cannula has been followed by distress, which has necessitated replacement. The girl is now 12 years of age; she is well built, active physically and mentally. A larger tube than that seen in the illustration has now been fitted, enabling her to breathe freely and to speak easily with the help of an inspiratory valve. She can carry on all activities except bathing, which would be dangerous if the neck was submerged.

referred to me where the condition was attributable to the previous use of diathermy in the area of the glottis.

Temporary or Permanent Tracheostomy: In many cases of severe stenosis opening the trachea is first required and should not be postponed too long. The numerous conditions calling for the performance of this operation vary considerably in prognosis. The most suitable are chronic inflammatory cases of the slow, lupoid type, but it may be required for relief of active laryngeal tuberculosis. It may subsequently be possible to abandon the tube if satisfactory healing takes place. Healing with or without local cautery treatment, may, however, be followed by contraction of the scar tissue and glottic stenosis.

Tracheostomy may be called for in cases of stenosis due to double abductor paralysis and here, again, isolated cases of recovery may occur; I can recall one due to temporary peripheral neuritis, apparently of syphilitic nature, although previous specific treatment had been given without immediate effect.

Only too many cases are still met with where a high tracheostomy has been performed; the first necessity in such a case is to replace the tube at a lower level, well below the first tracheal ring and the cricoid cartilage. Unfortunately, in too many of these cases irreparable damage has been done by cicatricial stenosis, by inflammatory fixation of the arytenoid cartilages, or by injudicious local interference.

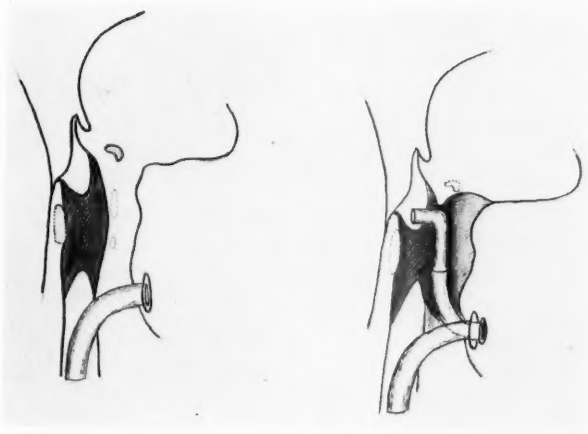
A subject of much debate must now be considered, and that is permanent tracheostomy. Owing to my good fortune in being the pupil and associate of Sir St. Clair Thomson, I have seen, in many and various conditions, the superior advantages of permanent tracheostomy compared with other treatments, and have been much impressed by the wisdom of this means of lasting relief. Theoretically there are many objections to the formation of a permanent stoma in the neck, but in practice it is very surprising to note what little disability is experienced, particularly when counterbalanced by the abundant airway and the preservation of voice. Children grow up healthy and well-developed (Fig. 1) and adults lead a normal existence, able to enjoy active sports and life, while women bear healthy children. As with "tubed" horses the one and only great risk is getting the neck submerged. (Fig. 2.)

One proviso must be made in claiming success for permanent tracheostomy, that some glottic space must be present for purposes of voice. The patient is provided with a hinged valve at the mouth

Fig. 3.—*Valve to Fit Tracheotomy Cannula.*—The hinged flap opens on inspiration and allows air to enter; it closes on expiration and allows the current of air to pass through the glottis for purposes of speech.

May 15, 1934 July 18, 1934 July 23, 1937

Fig. 4.—*Carcinoma of the Larynx Followed by Stenosis After Laryngofissure.* —May 15, 1934. Male patient, aged 53, with a carcinoma limited to the anterior region of the left vocal cord. This was removed by the laryngofissure route. Microscopical examination showed the growth to belong to Broder's Group 1. There was a satisfactory area of healthy tissue surrounding the growth. July 18, 1934. Appearance after operation. The aryteno-epiglottic fold on the operated side has been drawn inwards, while the arytenoid has moved somewhat forward. July 23, 1937. Three years later the glottis had become considerably narrowed owing to cicatricial contraction in the operation scar; there was no recurrence of growth. A tracheostomy cannula was inserted and was fitted with a valve for purposes of speech. (Case referred to me by Sir St. Clair Thomson.)



A *B*

Fig. 5.—*Complete Stenosis of Larynx.*—A woman, aged 31, had complete stenosis of the larynx, caused by a self-inflicted wound. Perichondritis had led to sloughing of the thyroid and cricoid cartilage. A fistula was established between the remains of the laryngeal aperture and the exterior by means of skin-flaps. The central part of the hyoid bone was removed. A groove was made in the front of the neck, leading from the fistula down to the tracheotomy opening. A hollow extension was made to connect the tracheal cannula to the fistula, with restoration of satisfactory speech in a loud whisper. *A.* The dark area represents the original lumen of the larynx replaced by scar tissue. The position of the cartilage is indicated by dotted lines. *B.* The groove and fistula are shown, with the tube extension in place.

of the tube, so designed that air can enter the lungs freely during inspiration, while part or all of it can escape through the glottis during expiration. (Fig. 3.) Patients dealt with in this manner include some who cannot separate their vocal cords because of fixation of the crico-arytenoid joints; others with permanent double abductor paralysis; others again with healed lupoid or tuberculous laryngitis; and a certain number with cicatricial stenosis following surgical traumatism, sometimes after operations for cancer of the larynx by the laryngo-fissure route (Fig. 4), or by lateral pharyngotomy. Finally, a few have stenosis from traumatic or suicidal wounds of the larynx.

January 5, 1932 December 14, 1932

Fig. 6.—*Stenosis of Larynx Caused by a Chondroma.*—The patient was an elderly lady with loss of voice, severe dyspnea and stridor. A large chondroma was blocking the larynx and also obstructing the esophagus. It was removed completely and, to repair the larynx, a flap of skin was turned in, leaving an open groove. Later the groove was converted into a tube and her voice and breathing powers were restored; the tracheostomy cannula, inserted as an emergency measure before the removal of the tumor, was no longer needed.

When dealing with healed malignant or tuberculous cases it would undoubtedly be unwise to attempt any active dilatation, for fear of promoting recurrence in a sensitive region.

Operations to Shift the Position of the Vocal Cords: Many attempts have been made, particularly in cases of double abductor paralysis, to restore the width of the glottis sufficiently to permit of easy respiration. I have the impression that such efforts may only result in an inadequate airway and leave a more defective voice, inferior in both respects to the permanent tracheostomy fitted with an inspiratory valve.

Amongst such operations to be mentioned are those of Rethi and Wittmaack for displacement of the cord outwards or downwards; that of Chevalier Jackson for removal of the cord by ventriculo-cordectomy; and also Hoover's method of submucous resection of the cord.

Nerve Anastomosis: In cases of double recurrent paralysis, anatomically successful operations have been performed by Colledge and Ballance and by Frazier for anastomosing the phrenic, or some other nerve, to the distal end of the injured recurrent laryngeal. The results do not appear to be satisfactory, possibly because of disordered co-ordination of the glottic movements.

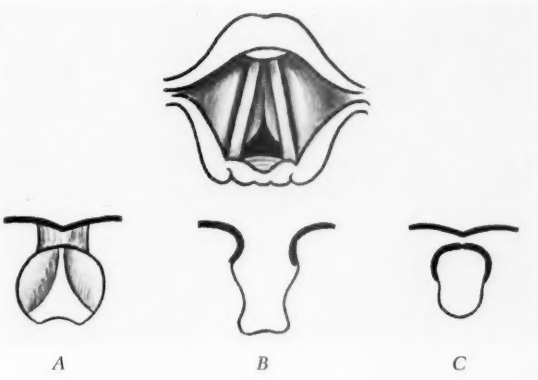


Fig. 7.—*Stenosis of Sub-Glottic Region.*—Female, aged 62, with history of huskiness for 20 years, getting worse, with recent stridor. A soft swelling of the posterior wall, causing obstruction below the glottis, was treated by a colleague with insertion of radium seeds, after the trachea had been opened. The swelling disappeared to a considerable extent, but stridor remained. The lumen of the trachea, just below the cricoid cartilage, was small and triangular in shape *(A)*. The trachea was split in the midline and skin-flaps were turned in and united to the tracheal mucosa *(B)*. The gutter so formed was later converted into a tube, with satisfactory restoration of the airway *(C)*. The tracheotomy cannula was no longer required, and the voice was restored to normal.

Continuous Dilatation: Many methods of this type have been described; some surgeons have made prolonged use of metallic obturators or doubled-up rubber tubes, others have employed moulds shaped to fit the larynx, while others again have inserted a hollow tube of vulcanite or rubber through which the patient might breathe. O'Dwyer's intubation tube may be employed.

Schmiegelow has described his method of holding a rubber tube in place by a transfixing peg of silver wire passed through the two alæ of the thyroid cartilage. In my practice such measures have given way in place of others to be described.

Laryngostomy with Prolonged Packing: This method, employed in some clinics, is tedious and painful. It aims at relining the restored laryngeal airway with scar tissue and epithelium, which spreads inwards from the muco-cutaneous margins. More rapid and effective methods appear to be available.

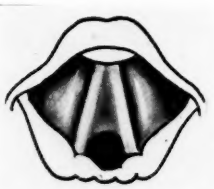


Fig. 8.—*Stenosis of Larynx Treated by the Skin-Grafting Method.*—The patient, a girl, aged 19, was in a motor accident and had her lower jaw fractured. Subsequently she suffered from dyspnea and hoarseness and a web was seen between the vocal cords; the laryngeal cartilages were fractured and the whole organ was shortened. Division with the cautery was attempted elsewhere. After transference to my care, laryngofissure was performed and the cords were separated and kept apart by an oiled silk bag covered with a skin graft. The result was good and the voice was improved, stridor was eliminated and there was no dyspnea on gentle exertion.

July 12, 1933 May 29, 1934

Fig. 9.—*Stenosis of the Larynx Treated by the Skin-grafting Method.*—In 1933 the patient (a man aged 49) suffered from laryngeal stridor. A swelling was seen below the glottis and this was treated with diathermic coagulation. Tracheotomy was called for to relieve the respiratory distress. When referred to me there was marked obstruction caused by adherance of the vocal cords. In 1934 the larynx was opened by the laryngofissure route; the vocal cords were adherent along two-thirds of their length, there being only a small posterior gap. The opposed surfaces were separated and a skin graft was inserted with the aid of Mr. T. Pomfret Kilner. It was kept in place by an oil-silk bag filled with ribbon gauze. The retaining bag was removed after seven days, and the tracheotomy tube eleven days later. The voice was much improved and there was no stridor or dyspnea on gentle exertion. The cords remained separate and free along their whole length after one subsequent dilatation.

Plastic Operations with the Use of Skin Flaps: I have employed this method in a few cases with some success, either in restoring the power of speech or, in addition, the ability to breathe without a tracheostomy. In one patient there had been extensive perichondritis of the laryngeal cartilages following a self-inflicted wound; the larynx as a tube no longer existed but was replaced by a mass of scar tissue (Fig. 5-A). The upper part of the epiglottis and the upper margins of the aryteno-epiglottic folds remained, but there was no communication between the pharynx and the trachea and, consequently, no voice.

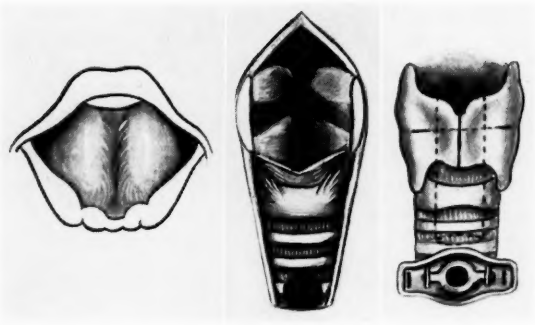
It was possible, after removal of the central part of the hyoid bone, to construct a fistula between the remains of the laryngeal aperture and the front of the neck by means of skin-flaps turned in to unite with the mucosa. A gutter was then constructed, leading from the fistula to the tracheal opening. A hollow, upward extension of the tracheostomy tube was used to connect the fistula with the trachea and thus to restore the power of speech, although a tracheostomy cannula was still required for inspiration (Fig. 5-B).

Amongst other cases I have dealt with by the use of skin-flaps is one of chondroma of the cricoid cartilage (Fig. 6) and another of cicatricial stenosis following irradiation of a subglottic neoplasm of uncertain nature (Fig. 7). In both of these a gutter was first formed, to be converted into a tube by dividing and turning in part of the skin flaps, with restoration of normal speech and respiration. It was possible to dispense entirely with any tube.

Continuous Dilatation with Skin Grafting: If the stenosis is in a region which may contract, the restored airway must be maintained for some weeks by the use of rubber sponges, Stent's material, rubber moulds, rubber tubing, or a silver extension attached to a tracheostomy tube. Having canalized the previously obstructed endo-larynx or subglottic region, the obturator is fitted. Instead of leaving the tract in which it lies to await the slow ingrowth of epithelium, the obturator is covered with a thin graft of skin, taken from the arm or leg. If rubber tubing is used it may be possible to dispense with a tracheotomy tube, but usually a cannula is retained until healing is complete.

This method has also been recommended by Arbuckle, Figi, and others, but its value needs emphasis, as it does not seem to have received the attention it deserves.

One girl, aged eighteen, operated on by myself, had not spoken for fifteen years because of complete subglottic stenosis, acquired at



A B C

Fig. 10.—*Complete Stenosis of Larynx.*—A boy, aged 16, fell off his bicycle and bruised the larynx. Stridor was noticed immediately, followed by cough and hemoptysis. Six days later respiratory distress necessitated tracheotomy. When first seen by me, two months later, there was complete stenosis. An interval was allowed to elapse before operation, to allow all inflammatory changes to subside *(A)*. The larynx was then split through a median incision and the adherent cords were divided with a scalpel. Movement of the arytenoid cartilages was at once noticed *(B)*. A skin graft was inserted wrapped around a rubber tube, held in place by Schmieglow's method of transfixion with silver wire passed through both thyroid alæ *(C)*. There was considerable reactionary swelling. The larynx was reopened and the tube removed twelve days later; the skin graft was in good position and firmly adherent. Subsequent progress was uneventful, with the return of a useful voice and a free airway. The tracheotomy cannula was corked 15 days after the operation and removed three or four days later. The figures show the larynx viewed from above, the larynx opened, with adherent areas, and the retaining tube in position.

the age of three, consequent on treatment for multiple laryngeal papillomata. The restoration of the subglottic lumen with skin-grafting, maintained in position by an extension upwards of the tracheal cannula, allowed her to speak without any real difficulty by the use of a flap valve. As no air had passed through the larynx all these years, the organ was of infantile size and insufficient for respiration, so that she could not dispense with the cannula.

Chevalier Jackson has referred to growth of the larynx as an important factor in restoring the airway; but in two or three cases in my experience, prolonged obstruction appeared to have arrested growth.

Skin Grafting with Temporary Fixation: Three or four cases have come to me with partial or complete stenosis following falls on the larynx; in each of them the vocal cords adhered together over a wide extent. It was possible, by employing the laryngo-fissure route, to separate the cords accurately and to insert a thin skin graft. I have used for retention either a bag of oiled silk packed with ribbon gauze, an obturator of Stent's material, or a simple rubber tube; I have found the latter to be the most efficient. The obturator or tube is left in place for a week or ten days.

One patient had considerable shortening of the glottis due to fracture of the thyroid and cricoid cartilages, but was enabled to speak well; she has no permanent tracheostomy, as the airway is adequate, and she can exert herself, after this treatment (Fig. 8).

Another patient had only a small triangular gap at the posterior end of the glottis; in him the vocal cords were permanently made free and restored to their original length (Fig. 9). A boy, recently dealt with, was voiceless owing to complete stenosis at the level of the cords (Fig. 10). The results of skin grafting, after laryngo-fissure, appear to be perfect in his case. The retaining tube was removed after twelve days and the tracheostomy cannula two weeks later.

In cases limited to the endo-larynx there is no marked tendency to contraction after operation and all goes well if the raw surfaces are prevented from reuniting. The application of a skin graft is so efficient a means of attaining this end that the general adoption of this method would appear to be desirable in suitable cases. The short period during which an obturator must be worn and the absence of prolonged after-treatment are advantages which should not be ignored.

This remark does not apply with equal force to stenosis of the trachea, or to laryngeal cases in which there has been much destruction of the cartilages.

The tendency to collapse of the re-formed tube then necessitates the continuation of the dilatation by the wearing of an obturator for some months.

NOTE—I wish to express my indebtedness to Sir St. Clair Thomson for his advice on the choice of methods and also for his kindness in reading and correcting this paper.

LXXIV

THE TREATMENT OF ACUTE NASAL ACCESSORY SINUS DISEASE

A. C. Furstenberg, M.D.

Ann Arbor, Michigan

How do you treat acute infections of the nasal accessory sinuses?

Such is the question frequently asked by our students of postgraduate otolaryngology. It arises from the mass of confusion regarding this problem; first, that there is little unanimity of opinion among the members of our profession; second, that treatments advocated today are changed tomorrow; third, that the proponents of specific therapeutic measures do not always practice what they recommend; and finally, that there is an appalling lack of sustained conviction on the subject of sinus disease in general.

Those who have expounded their own opinions have often been looked upon contemptuously by the students of different thought, so that it requires a little courage to pose one's personal views on this abstruse subject. Particularly is this true when we realize that pervasive methods of treatment, advocated by authorities on the subject, often differ to an extreme degree in the physiological principles which govern the care of acute infections.

The only generalization, therefore, which seems accurate about the treatment of acute nasal accessory sinus disease is that an inordinate variety of methods are in practice and that no specific cure, either medicinal, surgical or biological, has yet been discovered.

Perhaps I may be able to shed a little light on this subject by dealing particularly with some things that should not be done in the treatment of acute infections of the sinuses. It is true that to dwell on the negative phases of a subject is to admit one's lack of positive knowledge; nevertheless, there is much in some of our present therapeutic measures that is reprehensible, and the contention that there are things that should not be done can be supported by both clinical and laboratory experimentation.

And now let us answer the question: How do you treat acute infections of the nasal accessory sinuses? and to the answer subjoin the clinical observations and laboratory data which, it is hoped, will lend validity to our claims.

For the past 30 years, in the Department of Otolaryngology in the University of Michigan, nothing other than most conservative measures have been employed. In the early days, Dr. R. Bishop Canfield opposed incisively the use of any operative procedures in acute inflammatory diseases of the nasal accessory sinuses, and we have not discovered any good reason for deviating from this principle. "Avoid bone work in the presence of an acute infection in the sinuses," has been a surgical axiom of long standing in our department. It is an immutable law enforced even to the extent of discouraging the practice of antrum lavage, either by needle puncture or through a cannula passed into the ostium in the middle meatus. The former is certain to traumatize bone, the latter probably accomplishes the same effect in 25 per cent to 50 per cent of cases where even the skilled operator may encounter difficulty in introducing the cannula through the normal passage and occasionally perforates the thin osseous wall of the middle meatus. The avoidance of surgical measures of even the most minor proportions is a practice to which we give sedulous adherence.

But, apart from our concern about the dangers of injuring bone in the presence of an acute inflammatory disease, every effort is made to avoid traumatizing the sensitive and acutely inflamed mucous membranes. The use of irritating solutions such as the silver salts, colloidal preparations, mercurochrome, etc., is condemned and the application of a thick cotton or gauze tampon, which may do mechanical injury to the mucous membrane, is not considered an acceptable method of procedure in our clinic. No chemical agent is used which may irritate the Schneiderian membrane and no mechanical methods such as suction, tamponade or surgical intervention which may injure the linings of the nose or osseous structures are employed. Our practice, which has been in operation for many years, is of such a conservative nature that we are not warranted in speaking of it as a specific method. It embraces only those orthodox principles which are more or less applicable to all forms of infections, notably: rest, promotion of drainage, relief of pain, hydration and nutritional support for the patient.

The sinus sufferer is put to bed in order that he may avoid fatigue and exposure to rapid changes of temperature. Morphine or codeine are administered freely and whenever necessary to allay pain.

There need be no apprehension about the liberal use of adequate doses of narcotics, since the illness is evanescent and sedation for the relief of pain will be required only for a period of several days. The question is frequently asked, how are we going to relieve the antrum sufferer unless we irrigate his sinus? To this question we can only reply that morphine in large enough doses, that, is, quantities which give the desired pharmacological action, is our best available antidote for pain. It serves its purpose well in other excruciatingly painful diseases such as angina pectoris, stone in the common duct and Dietel's crisis, and it will give equally good effects in tempering the pain of an acute infection of the maxillary sinus. Moreover, it has been a frequent experience that patients with an acute nasal accessory sinus disease usually present a pansinusitis and are victims of the pain produced by retained secretions in several of the other sinuses, at the same time suffering the general reactive effects common to septic processes no matter where they may occur in the body. Obviously a local attack upon the antrum under such conditions, even if it accomplishes the purpose for which it is designed, can be expected to influence but slightly the pain and general suffering of such a widespread inflammatory process.

Heat or cold applied to the sinus regions occasionally exert a beneficial effect in relieving pain, although it is doubtful that either extreme of temperature possesses any potent values of a therapeutic nature in this disease. Steam inhalations (unmedicated) are frequently employed and ephedrine sulphate, 3 per cent solution in water (not the solutions in oil), is gently sprayed into the nose or dropped into the nasal cavities with the patient's head in the Proetz position. The latter measures are the only ones used by us for their shrinking effects upon the nasal mucous membranes.

The irritating influences of menthol, camphor or other medicinal agents in the steam and the volatile oils frequently used in combination with ephedrine, are avoided for reasons which will be presented in a discussion of the experimental work offered in support of the measures above advocated. Careful attention is given to the nutritional requirements of the patient and by the administration of copius quantities of fluids, every effort is made to keep the sinus sufferer well hydrated throughout the course of his illness.

The point of the foregoing is that most conservative means of combating an acute infection in the nasal sinuses are recommended; that nothing of a specific character is advocated, other than the assiduous avoidance of any measure, chemical or mechanical, that might be harmful to mucous membrane or bone which is already taxed to the limit of its natural powers in the fight against an acute infection.

You are not content, I am sure, to accept unchallenged, all of the above assertions. It therefore becomes necessary to present evidence in support of these claims. As previously stated, the basis for our convictions is twofold: first, clinical observations, and secondly, laboratory experimentation. Let us look into the former.

During the past 25 years many complications of acute nasal accessory sinus disease have come to our attention. They have ranged from more or less local processes, such as osteomyelitis of the bones of the face and skull, to systemic conditions of varying degrees of severity exerting their influence upon the heart, kidneys or other remote structures of the body. For example, a boy, aged 12 years, came to us suffering from a diffuse osteomyelitis of the skull which became manifest 10 days following an intranasal drainage of the left antrum. A doctor entered the clinic with a streptococcus septicemia which had developed two days following the needling and lavage of an acute maxillary sinus. A nurse consulted us because of symptoms of a brain abscess, which followed an attempt to enlarge the frontonasal duct for drainage of an acute frontal sinusitis. Another patient sought advice on account of an orbital cellulitis which promptly followed a partial exenteration of an ethmoidal labyrinth which harbored an acute inflammatory process.

At first these experiences did not elicit any particular concern on our part. They were regarded merely as the "mine-run" of complications that were prone to appear in any clinic where large numbers of upper respiratory infections are treated. However, a more careful inquiry into this situation revealed an appallingly large group of similar cases. Out of 58 cases of osteomyelitis of the skull, 42 presented a history of some previous surgical attack upon an acute nasal accessory sinus disease or an acute exacerbation of a chronic one; our records demonstrated many instances of orbital cellulitis developing subsequent to operations upon the ethmoidal labyrinth, and there appeared an amazing number of blood stream infections in patients who had been treated surgically for acute inflammatory diseases of the sinuses. But what was of greater significance to us was the observation that these complications did not occur in our hands. Without exception they were seen in individuals referred to the clinic on account of the complications from which they were suffering. It was an extraordinary fact that patients hospitalized by us in the early stages of an acute sinus disease, and subjected to our routine measures, did not number among the group which developed the serious sequellæ of acute nasal sinusitis.

To perpend this question which arose as a result of the above observations, namely, Why did not these serious complications occur in our experience? I undertook to make a brief clinical study, to which I have already alluded, by which it is hoped that some of the claims for conservative treatment will be substantiated. A series of 300 cases of acute nasal accessory sinus disease was studied in the University Health Service and in the hospital clinic. Each patient was subjected to a careful history, a clinical examination and a radiographic study of the sinuses. When the diagnosis was established as explicitly as these means would permit, the patient was hospitalized and treated in the conservative manner above described. In not a single instance was any surgical measure employed; not even needle puncture and lavage of the antrum, nor irrigation of the sinus through the ostium. The average period of hospitalization was one week, the first five days of which the patient was strictly confined to bed. All of these patients were returned to us for check-up examinations six months to two years following their discharge from our institution. Of these individuals 296 were asymptomatic; apparently in perfect health and clinically and radiographically free from any evidence of chronic nasal accessory sinus disease. Four individuals of this series, ranging between the ages of 18 and 23 years, presented unmistakable evidence of a chronic maxillary sinusitis. Twenty-six of the group had experienced one or more episodes of sinus disease following the attack which first brought them to our attention, 12 of whom had been treated by lavage of the antrum through a needle introduced beneath the inferior turbinate. The point that strikes most significantly is that the four cases of chronic maxillary sinusitis occurring in this group were found among those 12 who had been treated surgically during an attack of acute nasal accessory sinus disease subsequent to their experience in our Health Service. From this statistical study at least one fact is derived, namely, that out of 300 consecutive cases of acute nasal accessory sinus disease, four individuals appeared later with a chronic infection of the antrum. In each instance this observation was made in those persons who had been subjected to an admittedly minor surgical procedure.

To be sure, we must acknowledge the fallibility of statistical studies. We are open to derision if we speak succinctly of facts derived from a compilation of this character. Perhaps it is coincidence that has furnished the results of this brief study. It must be pointed out, too, that in this series we were dealing with young, healthy students ranging between the ages of 17 and 23 years, and that while under our jurisdiction we were able to put them to bed

and keep them confined, until complete resolution of the infective process had occurred. It would be invidious, too, not to point out that the four cases of chronic maxillary sinusitis occurred in individuals who had had one or more acute attacks following their first consultation with us. However, after taking into account the possibilities of error in diagnosis, the vagaries and fallacies of statistical computations, not to mention the whims and prejudices of observers, we are still forced to the conclusion that utmost conservatism as the treatment of acute nasal accessory sinusitis is worthy of avowed recommendation by the members of our profession.

In medical circles, one frequently exposes himself to ridicule and places himself in an ignominious position by adhering rigidly to stereotyped rules. It is not my wish to leave the impression that modifications of the above therapeutic measures are not occasionally in order. There can be no doubt of the value of antrum lavage in some cases of subacute maxillary sinusitis. In using this term subacute, I refer particularly to those sinuses that continue to drain a thick, purulent discharge for a period from three to six weeks following the subsidence of the acute fulminating infection. All of us have observed the satisfactory results derived from one or more irrigations of such a diseased sinus; but here again it is to be emphasized that the process to which reference is now made involves a stage intermediary to the acute and chronic disease. I need not point out again that my text deals specifically with the early acute phases of accessory sinusitis in which we encounter pain, discharge, sepsis and an edematous, inflamed mucous membrane. For this stage of the process, we adhere sedulously to conservative principles of treatment.

Reference has already been made to the laboratory data which may, in a measure, support our claims and furnish a newer concept in the treatment of infections involving the mucous membrane tracts of the body. We speak rather vaguely of the resistance of the patient and the virulence of the infection, but little success has been attained in the study and definition of these terms. Recently, however, some inspiring work has been conducted in our laboratory of Bacteriology under the leadership of Dr. W. J. Nungester on the effects of mucin on the virulence of pathogenic bacteria. He has produced unassailable proof that when bacteria are suspended in sterilized mucin and injected intraperitoneally, subcutaneously or into the lower air passages, they are so injurious to the animal host that it will frequently die from small sublethal doses. In other words, he has produced convincing and indisputable evidence that bacteria in the presence of mucin increase to an appreciable degree the virulence of the infection.

It would be superfluous to cite his numerous scientific observations. Suffice it to say that, in concluding one of his recent publications concerning the effect of mucin on bacteria, he makes the following comments: "The mechanism of the action is not yet understood. Mucin does not interfere with phagocytosis but does inhibit the bactericidal properties of phagocytic cells. It enables bacteria to survive in the body of the host for longer periods without diminution in number, or it may allow the organisms to increase in number and so result in the death of the animal. The viscosity and cohesive properties of the mucin appear to be important indices to the effectiveness of mucin on bacterial infections."

In connection with this work we have undertaken to study the effect of certain chemical and mechanical stimuli upon the production of mucin in the mucous membranes of the nose. Such preparations as argyrol and mercurochrome when dropped or sprayed into the nose cause a rapid and profuse secretion of mucin. Likewise, the slightest trauma of the nasal mucous membranes, even the gentle application of a cotton tampon, dry or saturated with sterile water, will activate the secretion of mucin; and the mere needle prick of the mucous membrane is a potent stimulus in activating, perhaps in a reflex manner, the mucous glands of the Schneiderian membrane. It was interesting to observe that any kind of irritant, chemical or mechanical, excited the secretion of mucin, the quantity of which was in direct proportion to the intensity of the stimulation.

From our recent studies, which have been in progress for a period of only 60 days, we have observed that irrigation with physiological salt solution is a potent method of dispelling the mucin. The latter is freely soluble in the salt solution and is disposed most effectively by the saline irrigation. If we are willing to accept the view that mucin, in the presence of bacteria, increases the virulence of the infection, and this claim has the fiat of leaders in the field of bacteriology, then the careful employment of saline nasal irrigations in coöperative adult patients may be a therapeutic suggestion of plausible value. This recommendation should be posed, however, solely on the assumption that the physician knows how the irrigation should be given and is fully aware of its mechanical dangers when injudiciously performed.

CONCLUSIONS AND COMMENTS

1. Our experience with acute nasal accessory sinus disease in the University Hospital, where many such conditions are seen each year, gives credence to the conviction that conservative measures

suffice in the vast majority of cases to bring about a successful resolution of the infective process.

2. The hazards of surgical attacks upon the walls of acutely infected sinuses, and risks of serious complications attending them, are herein emphasized.

3. A critical analysis of 300 consecutive cases of acute sinusitis would seem to indicate that surgical measures, even of a most conservative nature, are rarely indicated.

4. The effect of mucin upon the virulence of bacteria opens up a new and fascinating field of study. Our leaders of bacteriology have given authoritative support to the claims that when bacteria are suspended in a menstruum of mucin, they become most destructive invaders of the animal host. From these scientific observations there are, perhaps, lessons to be gained for the physician in the practice of rhinology.

UNIVERSITY HOSPITAL.

LXXV

MANIFESTATIONS OF ALLERGY IN THE EAR

MARVIN F. JONES, M.D.

NEW YORK

Allergy as a name has become a waste basket of such proportions as to be almost ludicrous. There is, however, a mechanism of the body which, when disturbed, produces many symptoms associated with the term allergy. Fluid balance in the tissues, the vasomotor sympathetic control, the endocrine system and disturbed metabolism has each its own part in producing the clinical changes of allergy. In attempting research on this subject, one is not faced with the problem of how it is possible to start, but rather with the problem of selecting a few of the most promising of a multitude of studies.

In discussing allergic manifestations as found in otology, it is first necessary to admit that little is known of allergy. The various tests, such as the contact, scratch and intradermal methods of diagnosis are fairly accurate for the inhalant group, but notably misleading when the offending substance is among the foods. Physical allergies have been cited, including those caused by changes in temperature, sun exposure and the effects of altitude and humidity. Absolute proof of an existing allergy is most difficult.

The personal statement of Ramêrez seems to place the subject on a more rational basis than any other I have heard. He said that the disturbance of the autonomic nervous mechanism causes various vascular reactions that are classed as allergic phenomena. Only a few of the symptoms arising from this disturbed mechanism are known and accepted. It is because of the unknown possibilities that we subject all obscure diseases, and a few of the classified diseases, to the question of their allergic origin.

The manifestations of allergy which may be recognized in the upper respiratory passages have been described and widely accepted. That allergic reactions involve the mucous membrane of the eustachian tube and tympanic cavity, as well as the nasal mucous membrane, has hardly received the consideration which such a plausible

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process suggests. By means of a nasopharyngoscopic examination, we may prove to ourselves that the visible part of the eustachian tube is subject to the same objective changes which have been considered characteristic of allergic changes in other nasal membranes. One observation which I have noted during nasopharyngoscopic examinations, and which observation I have not seen described elsewhere, is the character of the membrane on the posterior part of the septum. Strangely enough the peculiar appearance is not only present in nasal allergic conditions of long duration, but, when its presence is noted, there is a high percentage of patients who have complaints referable to the ear. I had hoped by this time to have a sufficient number of observations to make statistics of some value. Since the statistics are lacking, the observations may prove of value in your own future studies.

As the nasopharyngoscope is faced toward the posterior extremity of the nasal septum, an area anterior to the posterior border may be visualized. The diseased tissue under examination has the appearance of a fold of membrane overlying the septal mucous membrane and not unlike a false membrane. The color is a bluish gray and the surface is pitted. The condition frequently occurs bilaterally and occasionally is of sufficient proportions to partially obstruct the air passages.

In the nose we recognize a profuse thin, watery discharge as part of the symptomatology. We frequently notice a profuse watery discharge from the tympanum following a myringotomy. Nor is it strange that this should be so, since the eustachian tube which might furnish the only egress for fluid would be effectively blocked by the same process.

We have frequently inflated the middle ear cavity through the eustachian tubes and encountered fluid which sometimes persisted in spite of our best efforts to aspirate. The appearance of the membrana tympani in these patients may be without noticeable change. When we are able to aspirate this fluid the character seems the same as the nasal secretions caused by allergic attacks.

It has not been so long ago that "phenol and glycerin drops" were considered almost specific for painful ears. Perhaps with carefully selected cases we might find that the glycerin was sufficiently hygroscopic to reduce the non-infected fluid entrapped behind the tympanic membrane. Ichthyol and glycerin has a similar effect.

What is known of an allergy which might cause otological symptoms? There is ample proof that it is essentially a disturbance

of the vasomotor mechanism. Conditions such as angioneurotic edema, vasomotor rhinitis, conjunctival edema and the edema of the lining membranes of the sinuses, may be cited as examples. Disturbed fluid balance may be named as a result or possible cause. It is improbable that these recognized changes in the major membranes of the respiratory passages and the eyes would not also affect the membranes of another extension from the upper respiratory system, *i. e.*, the eustachian tube and the tympanic cavity.

The following case is chosen from a group to illustrate a probable acute allergic otitis media.

REPORT OF CASES

CASE 1.—B. J., aged seven, is a chocolate, nut and wheat sensitive patient. After ingestion of nuts Christmas day, he was seen to be rubbing his eyes vigorously. This was followed by rather profuse thin nasal discharge. The following morning at four, he began to rub his left ear. At eight o'clock he complained of stabbing pain. The drum at this time had lost the normal retraction and was flush with the malleus handle. There was some injection of the annulus and along the malleus handle, the remainder of the drum was pale. Fifteen per cent ichthyol and glycerin on a tampon was placed in the canal in close proximity to the drum. The boy was at some distance from the city, so he was placed in a machine for the trip to New York. The pain ceased on his way in at about eleven o'clock in the morning. That night the opposite ear was similarly involved. Both ears were normal in forty-eight hours.

Realizing that years of experience would make us slow to accept the idea that uninfected fluid *per se* could cause otalgia, the fact still remains that an enclosed infection would be expected to progress and not regress in the absence of drainage. On the other hand, an uninfected fluid might be eliminated by dehydration.

The following case is selected from a group diagnosed as allergic otitis interna:

CASE 2.—A farmer, the father of a nurse, had, for the past one and one-half years, been disabled by recurrent attacks of dizziness. The attacks occurred one or more times a week. It was necessary for him to have an attendant during this period. Numerous methods were tried to alleviate his dizziness. His daughter was a nurse visiting my clinic, and following the demonstration of a milk sensitive case she told me of her father and the amount of milk he consumed each day. Examination of this man was negative. The examination included a caloric test. The hearing tests were within normal limits. He was placed on a milk-free diet and for more than a three-year period he has been free of attacks. This is an illustrative case of allergic labyrinthitis and is one of a group of four similar milk sensitive cases.

It is impossible to say at the present time that these cases result from a disturbance of the end organ. They may be due to vascular insults affecting the vestibular nuclei.

The natural question arises regarding the validity of these cases being classed as allergic. Probably the best proof of their allergic origin is in the results obtained by treatment. Children, and adults also, who have been the victims of recurrent otitis, either of the middle or inner ear, are placed on a routine which eliminates most of the offending substances.

CASE 3.—Example of the results of this treatment is J. F., a boy aged eight years. Since he presents a rather typical picture and a satisfactory result, his case will be presented somewhat in detail.

Since birth, his history was punctuated by one illness after another. He was a difficult feeding case in infancy with continuous recurrent colds and otitis. His appearance at the first visit was that of an underweight child, nervous, apprehensive, dry skin, colorless and listless. He was a typically frail youngster. He was forced to drink his quart of milk a day, although he disliked it thoroughly. Eggs, bread and sweets had been given to him freely in order to build up his weight. He had no appetite but he did have frequent gastric disturbances. Constipation was the rule for which cathartics were given. Constipation alternated with diarrhea. He was the son of solicitous Jewish parents, whose main concern was to protect Jackie. The usual repeated tonsillectomies and adenoidectomies combined with nasal treatments, alternated with coughs, colds and earaches to make the boy's life miserable. The world for him was one continuous don't. He was placed on the following regime:

All pets, flowers, overstuffed furniture, heavy drapes and stuffed playthings were eliminated. The bed and pillows were of allergin-free material. All toilet preparations, such as powder, perfumes, scented soaps, toilet waters and lotions were removed from his room. His diet consisted of meat three times a day and included soluble and non-soluble fats. Pork was not included. Sweet potatoes, rice and soya beans were the vegetables permitted; stewed pears, prunes, grapefruit or lemonade the fruits. He was told to avoid taking milk or milk products, eggs, shell fish, chocolate, wheat or wheat products. The vitamin deficiencies were counteracted by the administration of the concentrates. Beef liver was included frequently in the diet.

The only local treatment was ephedrin sulphate one per cent in normal saline used as drops in his nose. At the start, four to five drops were placed in each side of his nose four times a day. The boy lay on his back in bed with a pillow under his shoulders. The head was tilted back until the point of his chin was over his external auditory meatus. He remained in this position five minutes. The period of this medication was approximately one month, and during this time, the number of treatments diminished gradually from four times to once a day. At the end of the month, an almost constant obliteration of the nasal passages caused by secretion and swelling had diminished, permitting him to breathe easily. At the beginning of the summer he was cautioned not to do any bathing except in his tub.

Various foods were added to his diet from time to time, attention being paid to those foods of valuable vitamin and mineral content but low in allergins. Any food to which he reacted three times was permanently removed.

The following winter he had two mild attacks of rhinitis and one attack of otitis. The summer was uneventful and he has had two colds with a recent mild

otitis which did not require a myringotomy during this winter now past. He is a normal boy in appearance and weight, and is a little too active for his parents.

Clinical evidence indicates that the phenomenon of allergy does affect the eustachian tube, the middle and the internal ear, although scientific proof is lacking. We do, however, know something of the manifestations as they occur in other parts of the body. Disturbance of the vasomotor or autonomic nervous system resulting in vasoconstriction or dilatation is accepted. Fluid imbalance in tissue does occur. The effect on gross body fluids, such as the cerebrospinal fluid, requires further investigation. There have been favorable clinical results following the alteration of the body chemistry, and it is reasonable to suppose that an aggravation of symptoms could result from the same chemical imbalance. Adrenalin acts specifically in allergic conditions, so we may also add that at least the suprarenal gland is a part of the endocrine system involved. These various subjects concerned in the phenomena of allergy may seem disconnected but further study shows them to be closely associated. Unfortunately, the very subjects involved are practically unexplored as far as definite tabulated information has been recorded.

In a symposium conducted by L. W. Dean before the Mid-Western Section of this Society, January 15th, 1936, Bronfenbrenner[1] made the following statement: "Immunology is not an exact science. Even the most fundamental concepts in immunology are only hypotheses, and in many instances these hypotheses are not accepted by all workers in the field. This is particularly true with respect to present-day understanding of the concept of allergy." I believe the statement is still true today. Since one guess is as good as another, a discussion of hypotheses here may not be out of order.

There are certain clinical and laboratory experiments to indicate that pressure influences both the static and acoustic labyrinth. Since I have not the courage to present the hypotheses on which we have been working for two years regarding the acoustic labyrinth, I will omit this phase and deal with the static labyrinth.

The first evidence that pressure may affect the terminal sense organs or cristae is found in the test for a fistula. In doing this test, air pressure is increased or diminished by pressure or suction from a Politzer bag connected to a rubber tip tightly fitted into the external auditory canal. If a fistula is present, nystagmus usually results. According to physical laws, the pressure applied to the fistulous opening is exerted equally in all directions. Therefore, any fluid which fills the bony capsule of the labyrinth would not be forced to flow

in any particular direction, but would be subjected to an increased pressure in all directions.

Second, we have all seen the resultant nystagmus following inflation of the eustachian tube. Here the tympanic cavity may be subjected to increase in pressure and if the secondary tympanic membrane follows the known deviations of the tympanic membrane proper, its flaccidity would easily permit unusual fluctuations in air pressure to be transmitted to its underlying fluid.

The third observation is perhaps a modification of the foregoing, in that the vestibular symptoms may be relieved by inflation which reduces a negative pressure in the middle ear cavity.

The fourth bit of evidence is furnished by the classical caloric test. Physics teaches us that the expansion and contraction of any fluid is effected promptly by heat and cold. The possibility of the labyrinthine fluid being caused to expand or contract by this classic test and thus causing change in pressure is at least interesting to contemplate.

As a fifth consideration, we might view our rotation tests in the light of centrifugal force. When the patient is rotated in the usual erect position, the normal reactions are of a mild character. However, when the patient is rotated with the body bent forward, the head resting on a metal support, the reaction in the same individual will be more severe. In the first instance, the radius from the semicircular canals to the center of rotation is much less than it is in the second position. Provided the speed of rotation is the same, the centrifugal force in the second instance would be much greater. The fluid pressure would likewise be increased.

The second phase of this hypothesis deals with fluid changes and their participation in pressure changes within the bony labyrinthine capsule.

The two fluids are the perilymph and the endolymph. The perilymph, of necessity, must be cerebrospinal fluid or a moderately filtered modification, since they are intimately associated through the cochlear aqueduct. The cerebrospinal fluid as contained in the subarachnoid area is in direct communication with the ventricular fluid through the foramina of Luschka and Magendie. The supply of this fluid is probably from the choroid plexus. All cerebrospinal fluid is subjected to constant changes in pressure. This pressure must, of necessity, be transmitted to the fluid of the labyrinthine capsule.

The endolymphatic fluid is in a closed membranous sack which also extends beyond the confines of the bony capsule through the endolymphatic duct to end in an extradural pouch, the endolymphatic sac. The origin of this fluid is proposed by Shambaugh[2] as being through the stria vascularis.

There is no known communication between the endolymphatic fluid and the perilymphatic fluid. It is not known whether their chemical constituents are the same. Since there are chemical substances in the cerebrospinal fluid, and since fluids having certain chemical compositions possess a potential osmotic pressure if separated by a permeable membrane from a fluid of different chemical composition, we are immediately impressed by the potential pressure changes in the adjacent labyrinth fluids.

Intracranial pressure can be changed by chemicals, including sodium chloride. Does it not appear that the elimination of sodium chloride, which has been so successfully used by Furstenberg,[3] may act by correcting a fluid pressure imbalance? Foldes[4] has written a complete text on the subject of water balance which will add value to this hypothesis.

All this seems rather far afield when we revert to our subject of allergy, but in pulling together the loose ends we have these points to consider: Can pressure cause labyrinthine disturbance? Does the autonomic nervous system control the vessels of the stria vascularis? Have the endolymph and perilymph got to be isotonic to maintain normal function in the static labyrinth? Can osmosis occur through the membranous labyrinth between the endolymphatic and perilymphatic fluids?

If these questions can be answered in the affirmative, we may rationalize our several methods of treatment to combat the fluid changes, vasomotor disturbance and endocrinopathy observed in the allergic phenomena.

121 EAST 60TH STREET.

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LXXVI

THE DEFICIENCY REACTION IN THE NOSE

E. R. Hargett, M.D.

Springfield, Ohio

A perusal of the literature on atrophic rhinitis does not disclose any universal theory as to its nature or its treatment. There is disagreement over the question whether ozena and atrophic rhinitis are the same or different, and whether the treatment of the two, if they be two, is the same or different.[1, 2] In attempting to clarify the issue, I would first state a few cases that have come under my observation, and a few of the facts that are known about the entity under discussion, keeping in mind that any rational theory or treatment must encompass the whole picture in a simple and reasonable way.

REPORT OF A CASE

Case 1.—F. B., a white male, age 33, had a severe blow on the nose five years ago. The acute swelling subsided, and the soreness disappeared without the benefit of medical attention, leaving no external deformity. But he has been unable to breathe through the left nostril since. It would be well to note here that he had absolutely no nasal complaints or subjective symptoms, nor discharge of any kind, nor odor, nor interference with his sense of smell, previously. Then, a few weeks after the injury, he began to notice a discharge of crusty material from his right nostril, which has gradually progressed until at the time of presentation, he regularly gets greenish-yellow crusts from his right nostril and his nasopharynx. He has on his own initiative attempted to wash these out with salt water. The other typical symptoms, with loss of smell, also developed. A submucous reaction was done, with removal of most of the columellar cartilage. Usual postoperative care was given, and in one month there were no signs of atrophic rhinitis in the right side whatever, and his sense of smell had returned. Incidentally, when the left side was viewed at operation, and at all later times, there were no signs of the entity there.

The case above is not unique[3] and many men have had similar ones; atrophic rhinitis often develops after intentional or accidental removal of parts or all of one or more of the turbinates; it is often in such cases unilateral. It is well to mention here one of the cardinal principles of intranasal surgery, that as much of the lining of the nose (not necessarily of the sinuses) as possible should be saved, and to criticize those operators who regard operative perforations of the septum lightly. Many operators alto tear the membrane from the middle turbinate, especially the posterior half, when doing intranasal

ethmoidectomies, and I have seen this produce atrophic rhinitis. It often occurs in children, although not often pronounced enough to notice until they are ten years of age or a little older. It sometimes runs in families, and has undoubtedly some hereditary possibilities. Also, I know that malnutrition is a factor in its etiology. It does not extend into the pharynx, except in rare instances, as pharyngitis sicca. It seems to tend to improve when the patient reaches and passes middle age. It is improved and sometimes cured by accidents or surgical procedures[4] (Wachsberger, Moritz[5, 6]), reducing the amount of air passing through the nares. Some observers believe atrophic rhinitis is due to improper balance of body fluid chemicals. However, these theories cannot account for all the facts known about the entity.[7] Cases can, of course, be cited to uphold all these statements, but this is unnecessary, as they have been repeatedly observed; the point is, any acceptable explanation of the cause of the condition must at the same time rationally and automatically account for all of the above manifestations, and any others that it might present.

Barnhill[8] states, in discussing the cause of atrophic rhinitis: "Undoubtedly mechanical pressure on the nasal mucosa is frequently one cause. * * * often, the pressure is due to crust formation. Thick mucous collects upon the turbinates, dries into crustlike caps over wide areas of mucous membrane and, by contraction, chokes off to some extent the blood supply to the entire nasal mucosa." This is without doubt true, and helps to explain why the crusts perpetuate themselves and impose a continuous exhausting irritation upon the glands, even after the original irritant, for example, a sinusitis, has disappeared.

He describes narrowing the nasal chambers by paraffin injections into the lower turbinate and the septum, and by cartilage transplants in the same localities.[9] These treatments are, of course, merely mechanical methods of cutting down the air stream, and any measure of success they may enjoy is due to this principle. But how much more efficient it is to temporarily completely block the entire current for as much of the twenty-four fraction as is necessary for control of the condition. Barnhill[8] also cites a case of atrophic rhinitis with crusts in a child of five years; as Hajek[10] says, this occurrence of the disease before the development of the sinuses and in children from whom the tonsils and adenoids have been cleanly removed, blasts most of the theories.

Many rhinologists and pathologists have remarked upon the decrease in size of the bony attachments of the turbinates in adult

cases of ozena; this fact is due to the embryonic and childhood basis of the condition in these patients, and follows the general orthopedic principles of lack of development, rarification and atrophy of the skeletal system. In much the same manner that trophic or unused muscle is resorbed, the elastic erectile tissue of the nasal mucous membrane disappears in these patients. In fact, wherever in this paper the nasal mucous glands are referred to, the whole secretory mechanism of the glands is to be understood as included; thus the elastic erectile tissue structurally supporting and physiologically functioning with the glands is to be included in the irritation-exhaustion cycle, and shares in the other characteristics of the condition. The whole capillary bed with its arterioles likewise is subject to these reactions, and is to be included in any consideration of the nasal mucosa as an organ.

Hajek[10] presents numerous cases, some unilateral, most of which showed sinus infection, or at least sinus secretion, but states that the real etiology of the condition is unknown. He does not go so far as Michel[11] and say that ozena is always accompanied by accessory sinus infection. Hajek's editorial note is of interest, in that it shows the status of the problem, and for the reader's refreshment, I quote: "In spite of the repeatedly emphasized views in the above discussion that the facts cited by me in no way prejudiced the etiology of ozena, I have found the statement disseminated almost everywhere throughout the literature that I am the chief supporter of the theory that ozena is caused by accessory sinus infection. I must most energetically protest against these statements. My view concerns the evaluation of the findings as presented. In a discussion with Grunwald[12] I have emphasized this as follows: From the existing conditions in an advanced case of ozena one can draw no definite conclusions in regard to the beginning of the process, and without the knowledge of this beginning, all of our assumptions are cheap hypotheses. They are much worse, since they divert us from the only certain means of promising results, and this is the study of rhinitis in early childhood. The simple consideration that most of the accessory sinuses in the earliest childhood, to which we must date back the beginning of ozena, are still not at all developed, while in advanced ozena they very frequently liberate the secretion alone, command us to exercise reserve in regard to drawing further conclusive deductions. The supposition is substantiated that before the discovery of the localized disease which was demonstrated in the further course, something else must have previously existed; either a localized disease in another form or a diffuse disease process. There

always remains the question to be answered: Why does the nasal mucous membrane, after being bathed in suppurative secretion, at one time show hypertrophy, as we have observed this in numerous accessory sinus affections, and at another time, atrophy. The answer to this question alone contains within itself a deep etiologic problem which up to the present time stands unsolved." The true answer goes back then even farther than the rhinitis of childhood, as he suggests; it lies in the embryonic placement pattern of the mucosal glands, and in their potential reserve as determined by their environmental, nutritional and inherited strength. If the pattern is thick and the fundamental reserve strength of the glands is ample, the nose will respond to being bathed in suppurative secretion by continued and chronic hypertrophy; if the placement-pattern is sparse, and the reserve strength small, a transient hypertrophy will take place, followed quickly by exhaustion and atrophy, with the cycle of protective crust formation, and then squamous epithelization for permanent protection. In defense of my presentation, I think it well to mention here that all physiologists and clinicians now agree that the heart, liver and pancreas have an inherited reserve that is modified and produced by many factors; the latter two organs being glandular, there is no physiological reason why the theory and fact of reserve strength should not apply to the nasal glands.

Ballenger,[13] in discussing the rationale of atrophic rhinitis, in the presence of sinus infection, states: "The secretions from the sinuses, more particularly the frontal, ethmoidal and sphenoidal, flow downward over the nasal membrane, where they dry, forming crusts." If this were true I should be able to follow a solid path of dried material back to the sinus infected; in fact, the crusts are often sporadically distributed. Again, many infected sinuses in atrophic rhinitis secrete so small an amount of material that it is difficult to find it, yet if his statement be true, the entire nose in some patients must be covered by thick crusts from these droplets in less than twenty-four hours. Many other false statements regarding this disease exist in the literature, mostly the result of trying to correlate an erroneous theory with clinical facts.

The title of this paper suggests what I believe to be such an explanation, *i. e.*, that it is basically a deficiency disease, and that amplification of this thesis will explain all of the manifestations and show the way to its cure, if possible, or explain why its cure is not possible. More accurately, then, atrophic rhinitis is a deficiency of the mucous glands of the nasal epithelium, either purely through a lack of an

adequate number of them, or through a lack of normal function of those present, if their number be normal, or a combination of both.

When the stratum corneum is removed from the skin, the moist layers beneath refuse to submit to drying and exude lymph and fibrin, which coagulates and dries, forming a protective coating, so that the cells below continue their function in a moist state. The same reaction occurs in the mucous membrane of the nose, or of any other part of the body, when its mucous glands fail to function adequately. The micro-organisms normally inhabiting the nose invade these scabs and produce the typical ozena. As this condition persists over a long period of time, a secondary defensive measure begins to act in the form of an attempt on the part of the mucous membrane to form a stratum corneum for itself. This process begins at the junction of the skin and mucous membrane at the opening of the nares, and gradually extends backward. Here we see why atrophic rhinitis is most severe, and most often seen in the posterior half of the nasal cavity. This also explains why the middle-aged and elderly have it so infrequently; that is, this cornifying process has had time to develop, and extend into the posterior part of the nose, giving the almost white lining, the almost completely dried up turbinates and decrease in amount of exudate seen in late cases of the condition. One can now see why the condition never extends into the mouth and seldom into the pharynx, with the large and copious salivary glands to keep these parts moist.

I know of nothing that will help the condition after cornification has taken place; in fact, these patients do not complain a great deal at that stage of the disease, because nature has more or less compensated, and the odor and discharge of crusts has ceased, these being the most objectionable features of the condition anyway. But in the years before cornification takes place, certainly these patients deserve some relief. My treatment, therefore, resolves itself into trying to lighten the load of the glands present, to restore them to normal function, and perchance to stimulate their proliferation. So if one side of the nose is closed through a deviation, the amount of air going through the other side is equalized, and thus the work of the glands, by a straightening of the septum. I save all membrane possible during operative procedures, to avoid overloading the other glands and avoid denuding the membrane from an area normally in the path of the strong air stream currents. Those operations which reduce the amount of air going through the nose by moving the lateral walls inward, thus reduce the burden of the glands, and some cures are effected.

Turner,[14] through Douglas Gurthie and Charles E. Scott, says, "The etiology of ozena (chronic atrophic rhinitis) is still uncertain." They quote Grunwald, that all cases are the result of local disease in one or other of the paranasal sinuses, and then state that this rule is not universal. They then state that, "It is now generally admitted that ozena is the terminal stage of chronic purulent rhinitis." They quote Adam, who contends that the more frequent removal of adenoids has reduced the incidence of ozena.

The matter of paranasal sinusitis preceding the disease coincides with our theroy of deficiency, in that the chronic infection so weakened the normal, or only slightly pathological, membrane until the glands were exhausted by continual overstimulation from an irritating discharge, and ceased or almost ceased to function; the membrane could not maintain its moist surface, so the crusts formed to protect it from drying as stated above, and I diagnose atrophic rhinitis preceded by sinusitis, or accompanied by sinusitis. The crusts in themselves are foreign bodies, and may as such perpetuate the irritation to the point of permanent exhaustion and in the end atrophy of the glands, thus perpetuating the deficiency and so the atrophic rhinitis, in the manner of a vicious circle. These types may sometimes be cured by the continual douching method of treatment as can be deduced from the above explanation. Of course, in a case combined with well-defined chronic sinusitis at the time seen, conservative treatment of the latter should be simultaneous; or even minor operative procedures that sacrifice no membrane may be used. In resistant cases of antral infection, the Caldwell-Luc procedure may have to be resorted to for cure of the sinusitis; this is not so pernicious, as only the nasal membrane over the window is sacrificed, the lining membrane of the antrum being relatively unimportant to the ozena per se, as it is not in direct line of the air currents and so not so strenuously concerned in the moisture function of the nose. Thus, if I cure the infection, and the vicious circle is not too old and thus advanced, the subsequent douching treatment may cure the ozena. But, unfortunately, cases are seldom seen at this opportune time. However, it constitutes a stone in the arch of the theory.

As for the contention of Adam concerning adenoidectomy quoted above, these facts may be true, but the explanation goes a little deeper. If infection of a sinus is secondary to adenoid infection, and/or if the adenoid is really infected, as shown by careful direct examination of these tissues, then the removal of these adenoids may clear the infection and remove the factor producing mucous gland deficiency. But again, if adenoids are removed from an other-

wise normal appearing nose in a child with familial nasal deficiency, the air flow through the nose may be too much for the remaining glands to moisten. Thus atrophic rhinitis, either localized or general, may result from the adenoidectomy, indirectly, presupposing the local factor present in the membrane. One must judge each case with these things in mind.

As with the other organs of the body, it seemed reasonable to believe that the condition of the glands could be improved by periods of rest. This was accomplished by placing cotton plugs in each or both nostrils. In working out my treatment by this means, it was found convenient to have the patient place these plugs in the nose at bedtime, and remove them the next morning, thus giving the membrane eight or more hours of rest out of the twenty-four. Patients soon become accustomed to the mouth breathing necessitated and make no complaints. In children, I explain the plan to the parent, and with their cooperation have had no difficulty in carrying out the treatment. A typical case is that of a negro girl, aged ten. Before treatment by my method, she had bi-weekly irrigations with various solutions, mostly saline, in the clinic. After wearing the plugs at night for six weeks, no more crusts appeared in the nose, and the case continued under control with the wearing of the plugs every other night.

REPORT OF A CASE

CASE 2.—Another of my cases was a young woman of 30 years, white, single, of moderate circumstances. She had suffered a severe cold in the head two years previously, which had never entirely disappeared from the left side. The discharge had at first been pus, but had later changed to crusts, greenish in color, and with a foul odor. Examination showed a typical atrophic rhinitis in the crusting stage in the left nostril. After cleaning out the crusts there were a few irritated points, but no actual secreting nor infected point, nor discharging sinus. Roentgenograms of the sinuses were normal. The right side of the nose was normal. The tonsils were cleanly out, and no adenoid tissue was present. The diagnosis was unilateral left atrophic rhinitis after injury by chronic sinusitis, the latter having subsided. The patient was instructed to come to the clinic for cleaning the crusts away every other day, and to wear the cotton plug in the left nostril constantly. She was also told to inhale partially and to exhale totally through the mouth when she could remember to do so. In two weeks no more crusts formed, and in another two weeks the condition was controlled by the wearing of the plug at night only, and no douching was necessary. The plug was then worn every other night, with no occurrence of the crusts. In this case the mucosa of the right nostril was strong enough to maintain its moist surface under the burden of an increased air current, but that of the left side was not equal to the insult of a severe chronic infection.

If the plugs are used while there is active sinus infection, the patient immediately feels worse, and this is an indication to begin

simultaneous treatment of the sinus, always being conservative at first. As soon as the sinus begins to improve, the plug treatment may be begun, and at this stage the patient will usually remark on the improvement. Of course if the adenoids are infected, they should be removed, but it is well to give pre-operative douches daily for about a week.

Treatment of atrophic rhinitis with radium has apparently had some success, but has not been widely accepted, and is unsatisfactory in most cases. Its effect is thought to be brought about by destruction of the secondary infection.[15] Toxoids have been found to be without benefit.[16] Local heat treatment has helped a few cases,[17] but no permanent results have been reported, and I believe that this improvement is due solely to transient increase in circulation in the nasal mucosa. Improvement and cures reported after simple douching I believe are due to the fact that such treatment replenishes the cellular fluids, cleans and moistens the surface, and by thus keeping the membrane moist for a short time affords it some rest periods. The irritation-exhaustion theory links up all these cases in a reasonable manner. In fact, the principle is old, having been applied for some time by the urologist to the explanation and treatment of impotence.

Mortimer and others[18] suggest that the disease is due to some dyspituitary state, and use dihydroxy-oestrin in oil as a spray to the nasal mucosa. They find this stimulating to the conchal mucosa, and effective as a treatment. Their theory of atrophic rhinitis is inadequate in several ways; for instance it fails to explain why the disease develops after extensive nasal surgery, and does not answer Hajek's question as quoted above. Applying my theory to their results, one can see how the exhausted mucosa might be stimulated to further effort by this specific hormone acting on the elastic erectile tissue. I wish to add that further research is in progress in regard to the effect of various vitamins, of general nutrition, and this and other glandular products as adjuncts to the rest-plug treatment of atrophic rhinitis.

CONCLUSIONS

1. A theory explaining the entity atrophic rhinitis has been offered.

2. Selected cases can be adjusted by equalizing the burden of the glands present in the mucous membrane, or by granting them periods of rest.

First National Bank Building.

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LXXVII

AUDIOMETRIC STUDIES ON SCHOOL CHILDREN

V. Changes in Air Conduction Acuity after an Interval of Five Years, with Particular Reference to the Effect of Age and Sex

Antonio Ciocco*

Washington

One of the principal aims of an investigation undertaken by the U. S. Public Health Service on the hearing of school children has been the determination of the mode of onset and the degree of progression of hearing impairment in childhood. For this purpose periodical audiometric examinations have been made on the same group of children. In 1931 a group of 1,400 Washington, D. C., school children were given otoscopic and nose and throat examinations, and air and bone conduction hearing acuity tests with a Western Electric 2-A audiometer.[1, 2] About three years later it was possible to re-examine a number of these children in the same manner, and a report of the study was presented in this journal.[3] During 1936-1937 an attempt was made to examine as many of the original sample of 1,400 children as were still enrolled in the Washington schools. Altogether, among the children examined at this time, there were 552 who had been tested five years earlier. The hearing records of these children constitute the material of this paper, which has for its purpose the description of changes in air conduction acuity that apparently occurred during the interval of five years.

METHOD OF AUDIOGRAM CLASSIFICATION

In order to simplify the analysis, the audiograms of these children have been classified into the following five broad categories of types. Good hearing, slight loss for tones of speech range, marked loss for tones of speech range, slight high tone loss, marked high tone loss. This classification, which has been used in previous studies, divides the audiograms according to (1) the degree of auditory

*From Child Hygiene Investigations, Division of Public Health Methods, U. S. Public Health Service and the Department of Biology, The Johns Hopkins School of Hygiene and Public Health.

acuity, and (2) the two major types of hearing loss: loss of perception limited to high tones only and that which involves all tones and consequently, to a greater degree, the ability to understand speech.

The categories are defined as follows:

1. *Good hearing:* All tones tested, 64 to 8192 double vibrations (d.v.), heard at an intensity equal to or less than 20 decibels (db).

2. *Slight loss for tones of the speech range:* One or more of the tones of the low and middle range, up to and including 1024 d.v., heard only at from 20 to 35 db.; higher tones may have been heard either within the range of good hearing or within the limits stated here.

3. *Marked loss for tones of the speech range:* One or more of the tones of the low and middle range heard only at 35 db. or above; the higher tones may have been heard at any intensity.

4. *Slight high tone loss:* Tones of 2048, 4096 or 8192 d.v. heard only between 20 and 35 db.; all other tones heard at an intensity equal to or less than 20 db.

5. *Marked high tone loss:* Tones of 2048, 4096 or 8192 d.v. heard only at 35 db. or above; all other tones heard as in 1.

From the audiograms obtained at the two examinations of these children the mean auditory thresholds of the ears classified according to the above scheme have been computed and are presented in Table I to illustrate this classification.

CHANGES IN TYPE OF AUDIOGRAMS

When the hearing in both ears of each of the 552 children is classified in the manner described above, the auditory acuity at the first and at the examination five years later is found to be as shown in Table II. For this tabulation, the children are classified according to the ear which has the greatest loss of perception. Thus, under the heading of marked loss for tones of the speech range in one or both ears, are included children in whom one ear possesses good hearing as well as children in whom one ear demonstrates a slight loss for tones of the speech range. Similarly, under the heading of marked high tone loss in one or the other ear are included children with one ear having either good hearing or a slight high tone loss.

From Table II the following facts seem noteworthy:

1. During the five-year interval between tests, the number of children with some form of hearing impairment more than doubled.*

2. During this same interval, high tone loss is the form of impairment which developed with the greatest relative frequency; the number of children with either slight or marked high tone loss more than tripled.

3. The number of children found with impairment for tones of the speech range also increased, but not to the same extent as the increase noted in the case of high tone loss. At the last examination the number of children with slight loss for tones of the speech range is higher by only 20 per cent than that found at the first examination, while the number with a marked loss increased by only 28 per cent.

A clearer view of the direction of changes in audiogram type and in auditory acuity is obtained when the data in Table II are considered together with those given in Table III. This table presents the number of ears having each type of audiogram as observed at the first and at the last examination. From the two tables the following seem to be the important facts:

1. Ears with good hearing acquired high tone loss of a slight or marked degree more often than loss for tones of the speech range.

2. A marked degree of impairment, whether for high tones or for tones of the speech range, apparently did not often improve, at least to a degree sufficiently to class the audiogram in the categories defined as good hearing or slight loss. In particular it will be noted that 24 of the 25 ears with marked high tone loss conserved the same degree of impairment. The remaining ear acquired, in addition, loss of perception for the low and middle tones. Of the 50 ears with marked loss of perception for the tones of the speech range, fully 70 per cent retained the same type and degree of impairment, while 16 per cent improved sufficiently to be classed among the ears with good hearing and 10 per cent regained some perception of the low and middle tones but conserved a marked high tone loss. This relative stability in auditory acuity of the ears with the impairment de-

*It should be noted that the percentage (67.0 per cent) of children with good hearing found at the last examination differs very little from that (69.4 per cent) reported by the author[4] on hospitalized patients less than 20 years of age.

fined as marked is in agreement with the expectation based on a previous report.[3] In that study it was concluded that when the auditory thresholds for air conduction reached about 40 db., the probability of improvement was small, especially for the ears in which the bone conduction was also reduced.

3. The ears classified as having slight loss, when they did not improve or remain unchanged, developed, in the majority of cases, a marked impairment of the same type. That is, a slight high tone loss, for example, more often changed into a marked high tone loss than to a marked loss for tones of the speech range. Relative to its number, each of the types of impairment of slight degree contributed more cases to its corresponding type of marked degree than to the opposite type.

From all these facts, at least two important conclusions may be drawn. In the first place, it is seen that even an impairment of slight degree, which as defined here is usually detected only with the audiometer, can develop into a condition serious enough to be a handicap. It is seen that eight of the thirty-two ears with a slight loss for tones of the speech range developed a marked loss of the same type, and nine of the twenty ears with slight high tone loss acquired a marked loss either for high tones or for tones of the speech range. Moreover, from these data it can also be inferred that the development of a marked degree of impairment often takes place in a slow and gradual fashion. Although great caution must be used in interpreting data based on such small numbers, the fact must be considered that 30 per cent of the ears with a slight high tone loss acquired a marked high tone loss as compared with 5 per cent of the ears with good hearing at the first examination. Similarly, 25 per cent of the ears with a slight loss for tones of the speech range acquired a marked loss of the same type, while this occurred in only 2 per cent of the ears with good hearing at the first examination. These findings appear to suggest that if measures for the prevention of deafness are to be investigated, the detection of slight degrees of impairment may well be considered as a first step. This requires (1) an instrument capable of measuring with some precision the auditory acuity, and (2) repeated and careful testing of the hearing of children, especially those who demonstrate any degree of loss of perception.

In the second place, the repeated observations on these children point to the means of obtaining much needed information about the factors associated with high tone loss. At the first examination the

frequency of this type of impairment in this sample of children was inferior to that of impaired perception for tones of the speech range, but, during the five-year interval, cases of high tone loss developed to such an extent that the latter became the major type of impairment. Although the existence of high tone loss among children has already been reported,[2] it is met frequently only after the fourth decade, and has been thought due to various conditions from arteriosclerosis to syphilis.[4] The records used for the present study give no precise information regarding the state of health of the children, but both the frequency of this type of impairment and the fact that the children are actively engaged in school work suggest that the several disease conditions thought responsible for high tone loss are probably not considerably involved here, and that the fundamental conditions responsible for this type of impairment occur in childhood or even earlier.

RATE OF DEVELOPMENT OF HEARING IMPAIRMENT ACCORDING TO AGE

The 464 children with good hearing in both ears at the time of the first examination ranged in age from 7 to 15 years, with a mean age of 10.5 years. The frequency with which the children of each age acquired some form of hearing impairment during the five years between tests is shown in Table IV. From this table the following facts appear important:

1. There is no definite association between age and the frequency of development of impairment for the tones of the speech range. For this type of impairment there appears, however, to be some indication of a decrease in rate of development associated with increasing age.

2. Slight high tone loss developed with greater frequency among the older children than among the younger ones. Among the youngest children, aged 8 years and under at the first examination, 4 per cent developed this type of impairment, while among the oldest, 11 per cent acquired a slight high tone loss during the interval between tests.

3. The frequency of development of marked high tone loss apparently increases with age until the age of 10 and then decreases. At the youngest and oldest age groups are found the minimum percentages.

The regular increase with age of the frequency of development of slight high tone loss in contrast with the age trend for marked

high tone loss is of interest if at the moment not explicable. It would be expected that the age trend in the development of slight and of marked high tone loss should be similar. In view of the findings, however, it could be inferred that the rate of change from a slight to a marked high tone loss is not constant relative to age. Moreover, in this limited age range, it would seem possible that while the development of slight high tone loss may be a more or less continuous process with age, the development of marked high tone loss suggests that of a cyclical growth phenomenon. It is realized, of course, that the size of the sample (when divided into age classes) and the number of children with defective hearing is small, consequently the observations reported above have a limited value only and are not sufficient to warrant definite conclusions. On the other hand, it cannot be denied that these data reinforce the view already advanced regarding the importance of seeking in children the etiological factors of high tone loss.

SEX AND DEVELOPMENT OF IMPAIRMENT

Among the 464 children with good hearing at the first examination, 223 were boys and 241 were girls. The percentage of children of each sex who developed one or the other type of impairment is shown in Table V. It is seen that the development of impairment for tones of the speech range occurred with equal frequency in both sexes. However, the percentage of girls who retained good hearing is higher than that of boys. This is due to a higher incidence of marked high tone loss, especially among the boys; almost three times more boys than girls developed a marked high tone loss during the interval of five years. This is the main dissimilarity between the two sexes relative to the development of hearing defects; it is statistically significant and is independent of the slight age difference to be found.

At the first examination the mean age of the boys was 10.7 ± 0.1; of the girls it was 10.3 ± 0.1. The slightly younger age of the girls apparently did not exert any effect on the rate of acquisition of marked high tone loss. When the age distribution of the boys is made equal to that of the girls, the corrected percentage for the boys is found equal to 11.3, which is even slightly, although insignificantly, higher than the observed 10.8. Since the sex difference in the rate of acquisition of marked high tone loss is not annulled or even reduced by this correction, it means that the greater relative frequency of this hearing defect among the boys is not due to their higher mean age. It is to be concluded, then, that the devel-

opment of marked high tone loss occurs with greater frequency among males than among females. This fact rounds out the observations already reported on adults and on children.[2]

So far, and with the exception of age, sex is the only factor generally found associated with the development of high tone loss. It would seem, therefore, that a fruitful line for the investigation of high tone loss should have as its starting point the search for a more specific physiologic and general biologic meaning of the sex difference.

SUMMARY

Re-examination of the air conduction acuity of 552 Washington, D. C., school children after an interval of five years reveals:

1. In the group as a whole, the frequency of high tone loss increased by about 300 per cent, while that of impaired perception for the tones of the speech range increased only by about 20 to 30 per cent.

2. Of the cases with a slight degree of impairment, as defined, about one-fourth developed an impairment of greater degree, but of the same type, *i. e.*, either high tone loss or loss for tones of the speech range.

3. In the majority of cases, when a marked degree of impairment, as defined, was present at the first examination, the same was found five years later.

4. The development of impairment involving in particular the tones of the low and middle range does not seem to be related either with the sex of the child or with the age at the first examination.

5. The relative number of children who developed a slight high tone loss increased concurrently with the age of the children at the first examination. On the other hand, the percentage of the children who developed a marked high tone loss increased with age up to 10 years and decreased thereafter.

6. The percentage of boys who acquired a marked high tone loss during the interval was three times that of the girls. The remaining forms of auditory defects developed with about equal frequency in the two sexes.

TABLE I

MEAN AUDITORY THRESHOLDS FOR EARS WITH SPECIFIED TYPE OF AUDITORY ACUITY.

Air Conduction Acuity	Mean Threshold in db. for Each Auditory Frequency in d.v.								Number of Ears
	64	128	256	512	1024	2048	4096	8192	
Good hearing	1.5	3.1	7.1	10.9	5.4	2.7	4.8	2.9	1868
Slight loss for speech range	13.4	17.0	21.8	24.0	18.7	12.6	12.7	10.2	68
Marked loss for speech range	31.3	32.6	37.7	42.6	41.2	41.3	44.9	45.0	124
Slight loss for high tones	3.6	3.9	7.9	12.0	7.3	6.2	22.8	24.9	87
Marked loss for high tones	3.7	8.8	8.7	11.4	8.4	11.4	39.0	44.3	111

TABLE II

NUMBER OF CHILDREN WITH SPECIFIED TYPE OF AIR CONDUCTION ACUITY AT FIRST TEST AND AT LAST TEST, FIVE YEARS LATER.

Air Conduction Acuity	First Test		Last Test	
	Number of Children	Percent	Number of Children	Percent
Good hearing, both ears	464	84.1	370	67.0
Loss for tones of speech range, one or both ears:				
Slight	20	3.6	25	4.5
Marked	36	6.4	46	8.3
High tone loss, one or both ears:				
Slight	13	2.4	46	8.3
Marked	15	2.7	57	10.3
Slight loss, different types in the two ears	—	—	4	.7
Marked loss, different types in the two ears	2	.4	4	.7
Slight and marked loss of different types in the two ears	2	.4	—	—
Total	552	100.0	552	99.8

TABLE III

NUMBER OF EARS WITH SPECIFIED TYPE OF AIR CONDUCTION ACUITY AT FIRST TEST AND AT LAST TEST, FIVE YEARS LATER.

Air Conduction Acuity (First Test)	Air Conduction Acuity (Last Test)						
	Good Hearing	Loss for Tone of Speech Range		High Tone Loss		Total Number of Ears	Percent
		Slight	Marked	Slight	Marked		
Good hearing	829	26	22	55	45	977	88.5
Loss for tones of speech range							
Slight	14	7	8	3		32	2.9
Marked	8	1	35	1	5	50	4.5
High tone loss							
Slight	5	1	3	5	6	20	1.8
Marked			1		24	25	2.3
Total: Number of ears	856	35	69	64	80	1104	99.9
Percent	77.5	3.2	6.2	5.8	7.2	99.9	

TABLE IV

PERCENTAGE DISTRIBUTION OF TYPES OF AIR CONDUCTION ACUITY OBSERVED IN CHILDREN WHO HAD GOOD HEARING IN BOTH EARS AT FIRST TEST FIVE YEARS EARLIER.

Air Conduction Acuity (Last Test)	Age at First Test: 8 and Under	9	10	11	12 and Over
	Percentage of Children				
Good hearing, both ears	78.9	73.7	76.2	76.6	80.8
Loss for tones of speech range, one or both ears					
Slight	6.6	6.1	1.9	4.5	1.4
Marked	2.6	6.1	2.9	.9	.....
High tone loss, one or both ears					
Slight	3.9	8.1	8.6	9.9	11.0
Marked	5.3	6.1	9.5	8.1	5.5
Slight loss, different types in the two ears	1.3	.....	.....	.....	1.4
Marked loss, different types in the two ears	1.3	.....	1.0	.....	.....
Total	99.9	100.1	100.1	100.0	100.1
Number of children	76	99	105	111	73

TABLE V

TYPES OF AIR CONDUCTION ACUITY OBSERVED IN BOYS AND GIRLS WHO HAD GOOD HEARING IN BOTH EARS AT THE FIRST TEST FIVE YEARS EARLIER.

Air Conduction Acuity (Last Test)	Boys		Girls	
	Number	Percent	Number	Percent
Good hearing, both ears	160	71.7	197	81.7
Loss for tones of speech range, one or both ears				
Slight	9	4.0	10	4.1
Marked	6	2.7	6	2.5
High tone loss, one or both ears				
Slight	21	9.4	18	7.5
Marked	24	10.8	9	3.7
Slight loss, different types in the two ears	1	.4	1	.4
Marked loss, different types in the two ears	2	.9	.....	.....
Total	223	99.9	241	99.9

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LXXVIII

IV. THE PATHOGENESIS OF MÉNIÈRE'S DISEASE AND OF KINDRED CONDITIONS IN THE EAR AND THE REST OF THE BODY

S. H. Mygind, M.D.

AND

Dida Dederding, M.D.

Copenhagen

On the ground of our observations, experiments and therapeutical results, we think it justifiable to conclude that in case of Ménière's disease there is a question of disturbances of the water metabolism not only in the ear but also, with individually varying location, in greater or smaller parts of the entire organism. These disturbances have a tendency to periodic fluctuation, due to a series of external and internal factors, which seem to exert their action essentially through a partly local, partly general, vasomotor—particularly capillariomotor—dysfunction.

According to Starling,[1] it is assumed that the movement of liquid between blood vessels and tissues depends upon the interrelation between the hydrostatic and the colloido-osmotic pressure in the capillary blood. In the arterial part of the capillary loop the hydrostatic pressure is high, expelling the liquid from the blood, whereas in the venous part it is feeble, the colloido-osmotic pressure thus again withdrawing liquid from the tissues.

Starling's law explains why, all other things being equal, the hydrostatic pressure which is higher in the head of a person in a recumbent position than in an erect posture, during the night expells water into the tissues, so that the patients awake with impaired hearing, buzzing, giddiness, headache, vasomotor rhinitis, and swelling of the skin of the face, and that these phenomena, owing to the erect posture, disappear in the course of the day, with synchronous swelling of the legs.

And it is likewise easy to understand that a water test followed by dilution of the blood and, hence, a reduction of the colloido-osmotic pressure, brings about the escape of fluid from the blood ves-

sels, thus giving rise to all the phenomena observed during these experiments.

That the same phenomena fail to appear in normal persons placed under identical conditions is probably due to the circumstance that, in them, reflex actions in the normally acting vessels and capillaries prevent a change in the interrelation between the hydrostatic and the colloido-osmotic pressure. Such regulating reflex action are for instance required to prevent the labyrinth from reacting to every slight change of posture of the individual. In Ménière patients with their marked vasomotor dysfunction this regulation is probably disturbed, particularly in places presenting, for some reason or other, a locus minoris resistentiæ. How a previous lesion is able to bring about a local vasomotor dysfunction is illustrated by Bier's[2] well known observation of the seasick German academician, in whose pale face the cyanotic fencing-scars became very conspicuous.

The contrary initial phase, which we have observed during our salyrgan experiments, as well as during our other dehydrating procedures, is probably due to the sudden mobilization of a greater volume of liquid giving rise to excessive dilution of the blood and to a reduction of the colloido-osmotic pressure, so that the water leaves the bloodstream in places presenting a locus minoris resistentiæ with insufficient vascular regulation, i. e., in a predisposed labyrinth. Not until depletion by diuresis, perspiration and salivation and restoration of the usual water-binding efficiency of the tissues is the water withdrawn from the labyrinth again and the hearing improved with the appearance of thirst.

Under these circumstances water is deposited in the predisposed places, an edema appearing in the ear, particularly in the labyrinth, but also in the eustachian tube, in the brain or in the cerebral membranes in the mucous membranes of the nose and of the gastro-intestinal canal, in muscles, and last, not least, in the subcutaneous connective tissue, in the form of the so-called subcutaneous infiltrates.

As this is not a question of a lethal disease, nothing reliable is known about the pathological anatomy in Ménière's disease. In a case submitted to clinical examination by Berggren[3] and to histological examination by Gray, nothing abnormal was detected either in the labyrinth or anywhere else. Wittmaack[4] in two cases found concrements in the aqueduct of the cochlea (in the one case also a neurinoma in the basal winding), but they must probably be considered exceptional cases. Microscopy of the subcutaneous infiltrates

does not reveal anything abnormal. Hence we must assume that they are changes of so transient a nature that they are destroyed by our ordinary preparation methods, and are, perhaps, also difficult to distinguish from the normal picture. We must therefore endeavor to find other means of clearing up the pathological process. Consequently we shall give special attention to the subcutaneous infiltrates which we suppose to correspond to the other morbid changes in the ear.

It should here be noted that, if we have designated these changes as an edema, this is of a nature differing entirely from the edema known in nephritis and heart diseases. The edema proper is plastic, the marks of finger pressure persisting. As was previously mentioned, this is never the case in subcutaneous infiltrates, regardless of whatever size they may acquire. The edema proper preferably appears in parts where the connective tissue is loose, such as, for instance, on the back of hands and feet, over the malleoli, where the subcutaneous infiltrates either are absent or minimal. This is because the edema proper is due to an accumulation of liquid in the interstices between the cells, it is an inter- or extra-cellular edema. Hence, in case of subcutaneous infiltrates there must be a question of accumulation of liquid in the cells, an intracellular edema.

This intracellular edema and the symptoms following it may, of course disappear without any sequels if the eliciting cause is removed, though evidently only if it has not been of too long duration. Gradually the edema changes into secondary atrophic and fibrous alterations. The skin becomes coarse, thick, inelastic, with permanent venous ectasias. The subcutaneous tissue becomes tough and fibrous.

Now, however, we meet the difficulty that practically nothing is known of the intracellular edema thus postulated by us.

We have, therefore, requested the anatomist, professor Tibor Péterfi,[5] to carry out a series of investigations of this question. Already in 1934, we[6] suggested "that it is very possible that nutritional disturbances caused by capillary dysfunction and resulting, among other things, in deficient oxidation, might give rise to increased water-binding efficiency in the cells, with subsequent intercellular edema." This hypothesis now seems to be confirmed by a series of experiments on living cells and tissues.

If amebæ are brought into a hypotonic solution they will swell; if brought into hypertonic solution, they will shrink. These alterations of volume can be measured very exactly. But if the vitality

of the amebæ is in any way impaired, the swelling as well as the shrinking effect becomes markedly greater, the permeability is increased. This can be seen not only in case of mechanical lesion but also in case of insufficient nutrition and also under the biological influence of age.

If amebæ from a young, lively growing culture will increase their volume in distilled water with about 40 per cent, old individuals from a poorly nourished culture will under the same conditions swell about 100 per cent. But the far strongest effect of increased permeability is obtained by checking the respiratory metabolism. This can easily be done with carbon monoxide and hydrocyanic acid without killing the amebæ. When such choked organisms are brought into distilled water they will swell much more than normally respiring individuals. In young amebæ the swelling in distilled water is increased from 40 per cent, as said, to about 100 per cent. But if they are given oxygen over a sufficiently long time again, the swelling in distilled water is again reduced to the original value.

In living cultures of fibroblasts, where the alterations of volume cannot be measured as accurately as in amebæ, the microscopic preparations, on the other hand, very nicely illustrate the corresponding difference in swelling, in normal, old, mechanically injured and choked cells. (Figs. 1-4.)

Further laboratory investigations seem to suggest that similar processes under certain circumstances also take place in the labyrinth, particularly in the organ of Corti. Here we have to do with very delicate cells surrounded by fluid on all sides and with a very sensitive function. And to keep up this function independent of the alterations in the surrounding medium a minute osmotic regulation of the individual elements would seem a necessity.

Now Falbe-Hansen[7] has been able—in strict accordance with our clinical experiments—to produce an edema of the organ of Corti by poisoning guinea pigs with large intraperitoneal injections of distilled water. The swelling takes place particularly in the bodies of the Deiters' cells and in the hair cells. The changes are comparatively slight. More evident are the results of the opposite experiment, in which an injection of a hypertonic solution of sodium chlorid makes the whole organ shrink just as we see in cases of Wittmaack's so-called hypotonic degeneration. As this experiment corresponds to a dehydrating and improving effect by administering of salt in Ménière's disease, we also here are in accordance with our clinical

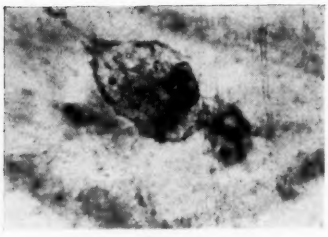

Fig. 1

Fibroblast punctured with a micro needle and immerged in distilled water, surrounding normal slender fibroblast (not sharply focussed). Magn. 1000x.

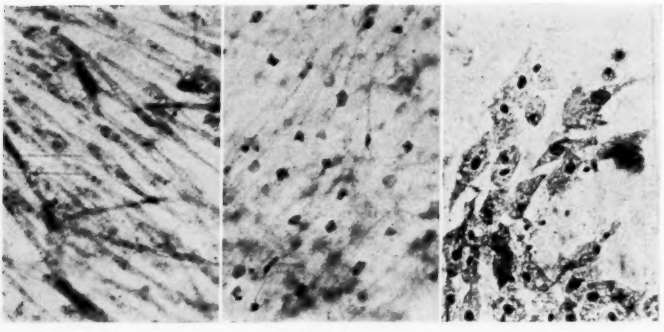

Fig. 2 Fig. 3 Fig. 4

Fibroblast cultures 3 days old, Magn. 100x. By Fig. 2, normal; by Fig. 3, treated 20 min. with distilled water; by Fig. 4, treated 30 min. with isoton cyanhydrogen and afterwards 20 min. with distilled water.

experience though in contradistinction to the conclusions of Fürstenberg and associates.

In these cases the organ of Corti has suffered on account of an osmosis-regulating demand beyond the physiological limit. In other cases the osmotic regulation may fail on account of an impairment of the vitality of the cells. Such an impaired vitality occurs in the stage between life and death and is the reason of the well known agonal changes so often seen in labyrinths, which have not been intra-

vitally fixed. These changes are not seen in any other part of the body. They are also particularly pronounced in the organ of Corti. In the beginning stage the agonal changes take the form of an intracellular swelling, which in its slightest degrees is very much like the swelling in water-poisoned animals described above. In animals treated with salicylates and quinin the agonal changes are much less pronounced (Falbe-Hansen) or even wanting just as in intravitally fixed animals. Correspondingly it was found by Péterfi that the edematizing influence of a hypotonic solution on living fibroblasts was also considerably checked by salicylates and quinin. Further, a series of not yet quite completed experiments on rabbits with salyrgan (Diuregan, Mersal) seem to prove the same preserving influence against agonal changes in the labyrinth, in due accordance with our clinical experience with the same drug.

We therefore think to have sufficient reason to suppose that in Ménière's disease we have to do with a cellular edema on account of a deficient vitality and an increased permeability of the cells of the organ of Corti. This deficient vitality is probably caused by a deficient vascular and particularly capillary function in a predisposed area. The predisposition is often evident from the history of the case and the otoscopic changes. The vascular deficiency is seen from the parallelism of the subcutaneous infiltrations with the vascular disturbances in the covering skin.

But why is the deafness a sound conducting affection and how are the characteristic feeling of pressure in the ear and the vestibular phenomena, particuarly the fistula symptom, explained?

The labyrinth forms a cavity with stiff walls, filled with fluid, a closed hydrostatic system ("closed box"). Even slight cellular edema will increase the content of the labyrinth and at the same time the endolabyrinthine pressure. The same will be the case of course if the production of the endolymph, eventually the perilymph, is increased (or its resorption checked), processes which probably also play an important part. Any increase of endolabyrinthine pressure will immediately bear on the fenestræ. This change of pressure gives rise to a fixation of the stapes from the inside which, just as the fixation from the outside that is known in middle ear affections, results in bass deafness, a sound conducting affection. It is true that this hypothesis in a certain degree is inconsistent with the result of an experiment performed by Hughson and Crowe[8] who, by plugging the fenestra rotunda, found an increase of the Wever and Bray effect, and not a decrease of it. However, the Wever and Bray effect certainly is not quite identical with hearing, and attempts at training

dogs with the conditioned reflex (E. Culler, G. Finch and E. Girden[9]) showed that the plugging of the fenestra rotunda reduced the hearing. Moreover, Szász[10] has reported experiments with pilocarpin injections on dogs, where he found an initially increasing and subsequently decreasing labyrinthine pressure, quite corresponding to the observation that pilocarpin administered to Ménière patients initially causes an impairment and subsequently an improvement of hearing.

Thus, whereas the fixation of the foot-plate causes bass deafness, eventually, as in other sound conducting affections, with a comparatively elongated bone conduction for a_1, we consider the bone conduction which, as compared to the air conduction, is not infrequently shortened and eventually quite absent, an expression of an outward dislocation of the foot-plate. This dislocation, which is a consequence of the increased endolabyrinthine pressure, causes the annular ligament to tighten (this probably is the cause of the sensation of pressure), and, in certain cases, to overtighten so that the foot-plate, when the pressure decreases again afterwards, is not quite restored to its proper position. The result is a lessened contact between the stapes and the frame of the fenestra, and decreased transfer of the vibrations of bone conduction.[11] The conditions met with in Ménière's disease are thus diametrically opposed to those of otosclerosis, where the isolating ligament is replaced by ideally conducting otosclerotic bone tissue. That certain cases present an abnormal mobility of the foot-plate in the fenestra ovalis because of an overtightened ligament is further evidenced by the not frequently occurring fistula symptom. The pressure feeling is probably the result of a stretching of the annular ligament.

Nor is it difficult to localize the vestibular symptoms in the labyrinth. They correspond exactly to what may be seen in case of labyrinth edema, which so frequently accompanies middle ear suppuration. Thus the most frequently occurring, homolateral spontaneous nystagmus, is explained as an increase of tonus due to the increased endolabyrinthine pressure exerting a compressing action on the sensory cells in the maculæ and cristæ. On the other hand, the heterolateral nystagmus, apart from the rare cases of hypo-irritability, is due to the intracellular pressure in the edematized sensory cells exceeding the extracellular pressure so that the sensory cells are not compressed but stretched. (We have endeavored[12] to utilize corresponding differences between intra- and extracellular pressure to explain the buzzing.) The fistula symptom is probably due to the previously described abnormal mobility of the foot-plate in the fenestra ovalis.

The apoplectiform attack is possibly due to the foot-plate not always accurately responding immediately to the fluctuations in the endolabyrinthine pressure but yielding in jerks, with momentary changes of pressure in the labyrinth following them. This is also suggested by the following observation of a Ménière patient during a dehydrating treatment:

At 9 a. m.

Whisper $\frac{aa}{0.30m.}$ Air cond. a_1 (norm. 25″) $\frac{4''}{6''}$ Bone cond. (10″) $\frac{0''}{0''}$

Lower limit $\frac{150 \text{ d. vbr.}}{150 \text{ d. vbr.}}$ Upper limit $\frac{14000 \text{ d. vbr.}}{14000 \text{ d. vbr.}}$

An air douche is administered without reliable influence on the hearing. Shortly afterwards the patient incurs a momentary, violent attack of giddiness. After this attack

At 9:30 a. m.

Whisper $\frac{aa}{20.00m.}$ Air cond. (norm. 25″) $\frac{4''}{19''}$ Bone cond. a_1 (10″) $\frac{0''}{5''}$

Lower limit $\frac{150 \text{ d. vbr.}}{36 \text{ d. vbr.}}$ Upper limit $\frac{14000 \text{ d. vbr.}}{16000 \text{ d. vbr.}}$

As is seen, the attack is brought on by an air douche and is accompanied by a very considerable improvement of hearing in the left ear, besides a greatly improved bone conduction, probably as a consequence of an improvement of contact between the foot-plate and the frame of the fenestra, as was previously mentioned.

This idea of the momentary attacks being due to sudden stapes movement is confirmed by the cessation of these attacks when the hearing is abolished, a consolation which such patients only too frequently have had to put up with. But as is shown by the function tests, a secondary stapes ankylosis very frequently is responsible for this excessive deafness.

Even though the acoustic and vestibular phenomena in a certain degree run parallel, it must be borne in mind that they must still present great symptomatic differences. It has repeatedly been emphasized that the vestibular symptoms, in contradistinction to the acoustic symptoms, do not only appear when the condition is exacerbated, but also when it improves. And contrary to the acoustic symptoms they tend to disappear, if only the morbid condition in the labyrinth remains unaltered. For the vestibular disturbances have the peculiarity of being gradually compensated so that their symptoms disappear. Hence even considerable labyrinthine changes can remain free from vestibular symptoms. This holds good in a still higher degree if the affection is equally marked on both sides.

Thus it is not the morbid process in itself, but the rate of its development or evolution and its greater or lesser degree of symmetry which is responsible for the most conspicuous symptom of Ménière's disease, namely, giddiness and nystagmus. Therefore the diagnosis depends so greatly on how often and how thoroughly we examine our patients and detect the actually somewhat accidental nystagmus abnormality. If we may say so, a person can very well suffer from Ménière's disease without presenting Ménièriform symptoms. But of course, the more markedly vasomotor the affection, the greater is the prospect of fluctuation in the patient's condition, hence the greater likelihood of a vestibular reaction. Now we understand also that there is some truth in the old assertion that Ménière's disease is a unilateral affection, for even though this is rare there will always be more marked vestibular symptoms, the greater the difference between the two ears.

Nor is there, as was previously mentioned, any sharp boundary between the typical Ménière's disease and the banal "middle ear catarrh," and tuba stenosis on the one side and that which is ordinarily termed neurolabyrinthitis on the other.

NOTE.—In our last paper, No. III, on page 772, we erroneously attributed the suggestion of a C-avitaminosis as cause of Ménière's disease to Göthlin instead of H. Öhnell.

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LXXIX

HYPERACUSIS*

H. B. Perlman, M.D.

Chicago

It has long been known that increased sensitivity to sound occurred occasionally in patients with paralysis of the facial nerve.[1] It has also been described in tabes, neurasthenia and amaurotic family idiocy. Krasznig,[2] in studying hearing disturbances in tabes, emphasized the need of differentiating clearly between an increase in the ability to hear sounds at threshold designated "oxyecoia" and an abnormality irritability or discomfort to sounds considerably above threshold designated "hyperesthesia dolorosa." The latter pertains to the findings in facial paralysis and tabes, while the former is probably more closely associated with the findings in neurasthenia and amaurotic family idiocy. It has generally been agreed, as stated in many neurology textbooks, that the increased sensitivity of sound observed in facial paralysis was due to paralysis of the stapedius muscle. These clinical observations were made, however, before the contraction of the stapedius muscle in the human was seen. A clear understanding of the function of the stapedius muscle did not exist at that time. Moreover, as late as 1924, Perekalin,[3] after reviewing the hearing disturbances in a series of cases of facial paralysis, concluded that:

(1) In all cases of paralysis of the stapedius muscle a hearing impairment was found, advancing the idea that the stapedius muscle acts to improve hearing.

(2) The stapedius muscle is of particular use in the perception of low tones, but its paralysis also produces a high tone loss.

(3) The impairment for the spoken voice and whispered voice is greater than for tuning forks, supporting the assumption that the stapedius is used more in hearing complicated sounds than simple pure tones. The conclusions of Perekalin on the function of the stapedius muscle were derived from a study of auditory function in patients with facial paralysis due to exposure to cold. The shortcoming of an analysis of such a group of patients is that not infrequently an associated neuritis of the eighth nerve exists.

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*Division of Otolaryngology, Department of Surgery, University of Chicago.

In 1929, Lüscher[4] first observed the contraction of the stapedius tendon in the human in response to acoustic stimuli, and found that a visible contraction did not occur at low intensity levels of sound, but definite above-threshold levels were required to make the muscle contract. He found that a sharp onset of the sound was necessary to obtain a brisk contraction, and further, that the tendon did not remain contracted for more than 70 seconds throughout a constant loud stimulus, since the interruption of the tone at any time beyond this did not produce any appreciable relaxation of the tendon.

In 1936, Lindsay, Kobrak and Perlman[5] analyzed the observations in a large number of individuals with visible contractions of the stapedius muscle and published the first quantitative studies of the sound intensities needed to obtain a just visible contraction of the muscle. They found that the threshold of the muscle contraction was about 65 db. above the threshold of hearing. As a result, it became clear that the stapedius muscle very likely did not enter into the mechanism of hearing sounds under 65 db. in intensity above threshold, but that its function was probably that of protection of the cochlea when sounds of sufficient intensity and of sudden onset reached the ear. An exhaustive study of the relation between the intensity of the sound stimulation, the latency period and the intensity of the contraction has been made on the rabbit's tensor tympani muscle by Lorente de No.[6]

Since the clarification of our knowledge of the function of the middle ear muscles, an opportunity to study quantitatively the effect of a paralysis of the stapedius muscle has presented itself in a patient with unilateral uncomplicated facial paralysis. A short report of the history and the results of various tests made upon the patient follows:

REPORT OF A CASE*

CASE 1.—A white male pharmacist, 45 years old, came into the neurological clinic on February 28, 1938, complaining of paralysis of the right side of his face. He stated that he had been well until a month ago when he developed a generalized head and chest cold from which he soon recovered. Two weeks later he noted that the sounds produced when dropping quarters and half dollars in his empty cash register were so loud as to cause an uncomfortable or annoying sensation in his right ear. The loud sounds as from a passing street car as well as the sounds produced by dropping coal into an empty coal pail, had a similar effect. The annoyance of a passing street car was relieved by turning his right ear away from it. The barking of his dog, when the patient was confined with him in the same room, also produced discomfort in the right ear. He resorted to the use of a cotton wool plug in that ear which relieved him of these annoying and uncomfortable acoustic sensations. There had been no pain or discharge in the

NOTE: Since this report was submitted two additional patients were studied in the manner described in the body of the report and similar findings were obtained.

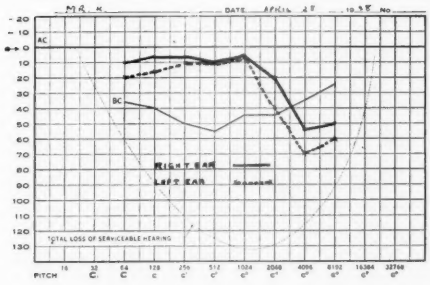

Fig. 1

ears. The left ear caused him no difficulty. Two days before coming to the clinic, and about twelve days after he first noted the irritability of the right ear to sudden loud sounds, he noted that he could not expectorate accurately. By the next morning he lost control of the entire right side of his face, including the ability to close his right eye. There was no definite history of exposure to cold or drafts. The irritability of the right ear to sudden sharp sounds persisted. He noted further that the spoken voice on the telephone at his right ear was "loud," "blaring," indistinct and uncomfortable. With the development of the complete facial paralysis, he noted that "sugar tasted like licorice" and "salt tastes bitter." There was no other pertinent history except that for about one year he had been drinking about 16 ounces of whiskey a day. The physical examination was essentially negative except for the complete facial paralysis. No other cranial nerves were found to be involved. The laboratory tests, including a Wassermann and Kahn test, were normal. He did not complain of deafness. Examination of the nose, nasopharynx and larynx was essentially negative. The drum membranes and auditory canals appeared to be within normal limits. The Weber test was not lateralized. The Rinne test was positive in both ears and the Schwabach test showed a very slight reduction for the 512 magnesium alloy tuning fork. The upper tone limit for the right ear was 16,000 by air and 18,000 by bone conduction, for the left ear 15,000 and 17,000 respectively, as tested with the monochord. He had no difficulty in determining the direction of a sound source and noted no difference in the quality of soft sounds heard in his right ear as compared to his left ear. He heard the spoken and whispered voice well in each ear. An examination of the auditory function with the Western Electric 2A audiometer (see Fig. 1) revealed that the threshold of hearing for frequencies between 64 and 1024 cycles was only slightly depressed or practically within normal limits. The audiogram revealed, however, an impairment of both ears for the frequencies from 2048 to 8192 cycles. The hearing impairment for high tones was more marked in the left ear or the ear opposite to the site of the facial paralysis.

The patient was then presented in ten decibel steps with tones up to 60 decibels above threshold and asked to compare the loudness of this tone in the right ear with that in the left. This was done by having the patient put the receiver of the audiometer to one ear and then to the other. Up to 60 decibels of sound intensity for frequencies from 128 to 2048 the patient could appreciate no difference in loudness between the two ears.

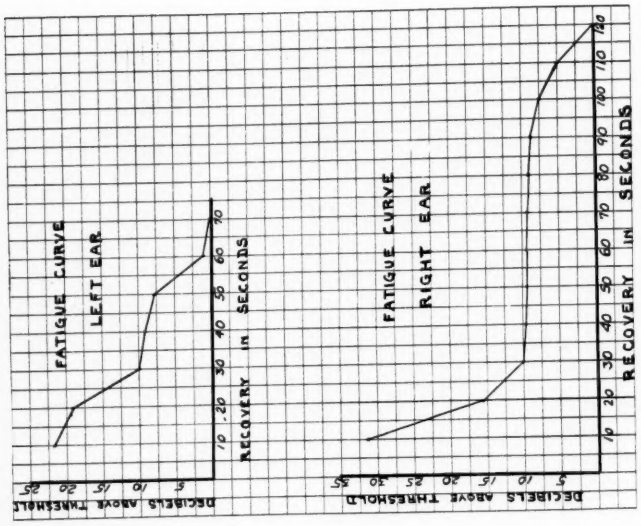
FATIGUE CURVE
LEFT EAR
DECIBELS ABOVE THRESHOLD
RECOVERY IN SECONDS
FATIGUE CURVE
RIGHT EAR
DECIBELS ABOVE THRESHOLD
RECOVERY IN SECONDS

Fig. 3

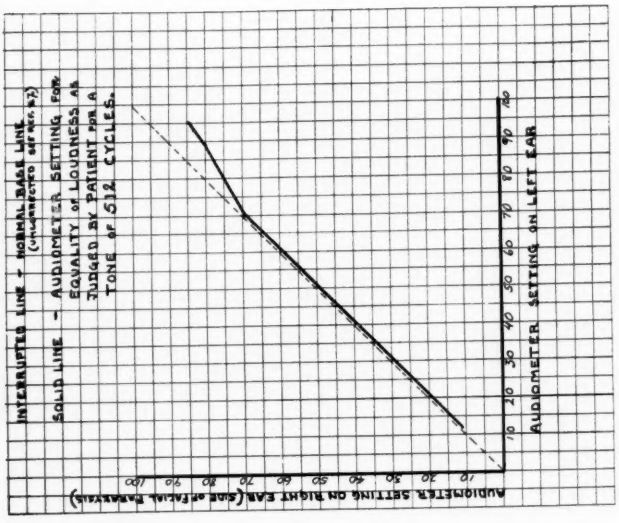
INTERRUPTED LINE = NORMAL BASE LINE
(OBTAINED BY AVERAGING 50 NORMAL EARS)
SOLID LINE - AUDIOMETER SETTING FOR EQUALITY OF LOUDNESS AS JUDGED BY PATIENT FOR A TONE OF 512 CYCLES.
AUDIOMETER SETTING ON RIGHT EAR (SENSE OF FACIAL PARALYSIS)
AUDIOMETER SETTING ON LEFT EAR

Fig. 2

He was then presented with the same frequencies at the maximum intensity produced by the 2A audiometer and a similar judgment of loudness made. At this level the patient noted definitely an increased loudness in the right ear over the left ear for the same frequencies. An increased loudness was first noted when the tones presented were 70 decibels above threshold. Loudness balance determinations at intensity levels between 70 decibels and the maximum of the instrument were made.

An example of the findings is that a tone of 512 cycles at 85 decibels in the right ear was appreciated by the patient to be equally loud with the same tone in the left ear at 95 decibels (see Fig. 2).

Further evidence to corroborate the findings in increased loudness was obtained by fatiguing the auditory mechanism on each side and determining the rate of recovery. This was done by exposing the patient's right ear to a sound of 1024 cycles at 100 decibels for two minutes, and then determining his threshold for this tone at ten second intervals thereafter until the threshold returned to the level present before the fatiguing tone was started. This was then repeated for the left ear. Curves (Fig. 3) indicate the decibel loss and recovery rates for the two ears. It is apparent that the fatigue was greater in the right ear than in the left and that the right ear required about twice as much time as the left to recover its normal threshold.

Another determination made was the degree of masking produced by a sound of 512 cycles at 80 decibels delivered to one ear upon a tone of 256 cycles delivered to the other ear. Two audiometers were used in this test. It was found that when the masking tone was at the right ear it masked a 256 tone of 27 decibels in the opposite ear. When the masking tone was at the left ear, the loudness was not sufficient to obliterate the same 27 decibel tone in the right ear.

The patient was next placed directly in front of a loud speaker at a distance of five feet with his ears equally distant from the sound source. With a special beat frequency audio oscillator a moderately loud sound of 1000 cycles was produced with a sudden onset and maintained. The patient observed that the sound seemed to "strike his right ear first" and was distinctly louder in that ear for about 45 seconds after which time, as the sound continued, he could determine no difference in loudness between his ears.

The fact that both ears were deafened for the frequencies between 2048 and 8192, as well as a slight bilateral shortening of bone conduction, speaks for a mild bilateral lesion of the perception apparatus, and may be excluded from the analysis except as this deafness affects the loudness judgment of the lower frequencies as shown by Steinberg and Gardner.[7] The sound intensity required to bring out a difference in loudness corresponds closely with the sound intensities required to produce a just visible contraction of the stapedius muscle in the human. It is therefore safe to assume that the loudness changes in the right ear are at least in part directly related to the inactivity of the muscle on that side, while in part due to the differences in hearing between the two ears. This inactivity permits greater excursions of the stapes than normally occurs when the muscle contraction dampens or impedes the motion of the stapes. Thus greater stimulation of the cochlea follows which is appreciated as greater loudness. The appreciation of equal loudness after 45 seconds exposure of the patient in a sound field is interpreted as resulting from some form of adaptation of the central nervous system. This removes the need of protection of the cochlea and soon results in a relaxation of the stapedius muscle as observed by us in patients with no paralysis and reported by Lüscher.

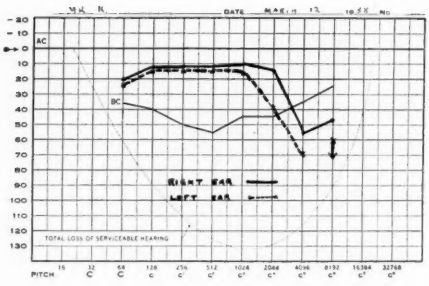

Fig. 4

Therefore after the above tone had continued for over 45 seconds it is probable that neither ear has an active contracted stapedius muscle and the auditory mechanism and central nervous system are equally adapted—resulting in a sensation of equal loudness. Evidence that this was not due to fatigue of the muscle of the unaffected ear is offered by the observation that a tone sounded immediately after the testing tone was again appreciated as louder in the affected ear.

This patient was again seen on April 28 or about 14 weeks after the onset of his illness. He stated that his facial paralysis had largely disappeared two weeks ago and at the same time he no longer experienced discomfort in his right ear when exposed to such sounds as were produced by dropping quarters and half dollars in his empty cash register, dropping coal in an empty coal pail, etc. Except for a slight return of his facial paralysis following a "cold" resulting in the drooping of the corner of his mouth on the right side his improvement has been maintained. He can close his right eye and describes but a slight remaining disturbance in taste —sugar tasting "metallic" on right side of tongue; vinegar and lemon taste normally. He no longer wears a cotton plug in his right ear and can use the telephone on the right ear as well as on the left. His hearing threshold curve as determined again with the Western Electric 2A audiometer is essentially as that found on the previous examination. (Fig. 4.) Now he detects no definite difference between the right and left ear in the loudness of sounds produced by the audiometer from ten decibels to maximum between the frequencies of 128 and 2048.

SUMMARY

The term "hyperacusis" should be more accurately differentiated into types. Two definite types are to be distinguished: First, a definite lowering of the threshold of hearing, or increased acuity which may be designated oxyecoia. Second, an abnormal discomfort caused by sounds above a certain intensity for which the term hyperesthesia dolorosa has been suggested. The latter type has been known to accompany facial paralysis and is considered as being due to paralysis of the stapedius muscle. A patient presenting this phenomenon

has been carefully investigated, as to hearing sensation for threshold and over-threshold sounds, auditory fatigue, masking and "adaptation."

The onset of the phenomenon occurred constantly at a definite intensity level for various frequencies, an intensity level which corresponds with the normal threshold for the stapedius muscle contraction. Also those sounds which the patient described as causing discomfort were of an intensity and sharpness to produce stapedius muscle contractions. Fatigue of the auditory mechanism was more marked in the affected ear and required a longer recovery period.

The phenomenon which might be described as "adaptation" could be demonstrated, namely, prolongation of an intense sound was accompanied by disappearance of the "discomfort" in a period of time closely corresponding to that in which the stapedius contraction is known (experimentally) to disappear, thus bringing about an equality of loudness in both ears. The study of these phenomena as afforded by this clinical case corroborates the findings previously recorded on the action of the stapedius muscle, and clarifies our conception of the part played by the stapedius muscle in the function of hearing.

950 East 59th Street.

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LXXX

THE HISTOPATHOLOGY OF APICITIS IN SUPPURATIONS OF THE PETROUS PYRAMID

ELMER A. FRIEDMAN, M.D.*

CHICAGO

In the past decade the subject of infection of the petrous pyramid has been a fertile field for investigation from its anatomical, pathological, clinical and surgical aspects.

Infection of the petrous apex is but one subdivision of petrositis. Almour has given us a detailed classification of petrosites on the basis of anatomic location. But from a consideration of the clinical symptoms it is not always possible to say in what portion of the petrous pyramid infection exists. Moreover, it has been repeatedly pointed out that there are no symptoms which are invariably associated with disease of the petrous pyramid. Friesner and Druss, in a histological study, found that in the majority of cases of suppuration in the petrous pyramid, the greatest expression of disease was at the posterosuperior margin of the petrous pyramid, between the superior semicircular canal and the internal auditory meatus. In only a few cases was the major expression of disease at the apex of the pyramid.

There are, however, certain facts which make infections of the apex of particular importance and of marked danger. There is a propinquity of vital structures such as internal carotid artery, pericarotid venous and nerve plexuses, cavernous sinus, abducens nerve and greater superficial petrosal nerve. The belief is widespread that there is a marked tendency toward spontaneous healing of infections of the petrous pyramid. Yet on purely anatomical grounds, when infection is once established in the apex, it must have more difficulty in drainage and less tendency to heal than infections elsewhere in the petrosa which are closer to middle ear and mastoid, and which therefore will tend to respond as the infection in these latter areas drains and heals. Finally, infections of the apex are of danger, as

*From the Department of Otolaryngology, Oscar Johnson Institute, Washington University Medical School.

Grateful acknowledgment is made to Dorothy Wolff, Ph.D., in whose laboratory the investigation was made, and to Professor L. W. Dean, Head of the Department.

has been so well stated by Almour in discussing lesions confined to the anterior perilabyrinth, since "These constitute a greater threat to life than do posterior petrosites, because, firstly, they are not demonstrable through the exposure afforded by the simple mastoidectomy, and, secondly, because of this, the search for the focus is often delayed beyond the safety point, due to the unwillingness of the surgeon to perform, or the patient to submit to the exposure of the tympanic cavity which is essential for the discovery of the lesion."

Although petrositis and apicitis are no longer considered rarities, yet it is doubtful whether it is realized that infections of the apex are so common.

In a routine study of temporal bones conducted in the Otological Research Laboratories of Washington University Medical School there have been found 20 specimens of apicitis out of 275 temporal bones. These constitute 7.2 per cent of the total number of specimens. By cases, there are 16 of 169, or 9.4 per cent of the total number of specimens studied. The following is a histopathlogical study of fifteen temporal bones, comprising eleven cases, four bilateral, which revealed infection of the apex. For the purpose of this study the apex is considered to be that portion of the petrous pyramid antero-medial to the cochlea.

Case No. 6379.—White male, age 6 years. Admitted January 30, 1936, died January 31, 1936. Entrance complaints of pain in right ear of one week's duration, although child had had a cold for a few weeks. Headache appeared and became worse, as did earache. Five days after onset a local physician noted bilateral otitis media and a temperature of 101 degrees. The next day the patient became comatose and had generalized convulsions. On entrance to Children's Hospital he was comatose in a continuous convulsive state. Discs blurred. Right drum full and thick, left drum not noted as abnormal. No neck rigidity or Kernig. Spinal fluid was slightly turbid, increased pressure, 98 cells, all lymphocytes. Spinal sugar 5, and on direct smear numerous gram positive diplococci were seen. Pneumococcus Type I on culture. Temperature from 40.5 to 41.8 degrees. He died 26 hours after admission.

Autopsy: Purulent bilateral otitis and purulent meningitis. No route of extension seen grossly.

Microscopic Observations: Bilateral apicitis.

Right Ear: Sections show an acute otitis and mastoiditis. The apex is extensively, though not completely, pneumatized with cellu-

lar tracts extending to the apex by the superior petrosal and hypotympanic routes. The diploic cells are in contact with the pneumatic cells, and both are the seat of a suppurative inflammation with much bone destruction. Infected apical contents are in contact with the dura of the middle fossa through a dehiscence, but the dura appears intact. There is pus, however, in the Gasserian ganglion. Multiple dehiscences are present in the carotid canal (Fig. 1); a subperiosteal carotid abscess is seen and a thrombophlebitis of the pericarotid venous plexus. Through a dehiscence, the posterior perilabyrinthine cells connect with the internal auditory meatus. Osteoclastic activity and pus are present throughout this fissure and a vein in its center is the seat of a phlebitis. A few pus cells are noted in the basal turn of the cochlea.

Left Ear: Similar to opposite side in anatomy and routes of extension to the apex. There is an acute otitis, mastoiditis and an acute apical infection in both air and marrow containing cells, but the process is relatively mild as compared with the opposite side. The Gasserian ganglion is infiltrated by pus cells, though a direct connection with the infected apical contents is not seen. The marrow at an inferior level is quite normal. Pus and hemorrhage are present in the pericarotid sheath and on this side also is present a pericarotid thrombophlebitis. Peribulbar cells contain pus as does the cochlear aqueduct.

Sphenoid: Mild infection present. Cavernous sinus on right a mass of pus.

Comment: Both ears reveal apical infection in air cells and diploic cells in a mixed type of apex with, however, pneumatic cell infection markedly predominant. The meningeal infection may be by vascular extension or secondary to abscess formation in the Gasserian ganglion. The process is undoubtedly older than one week, as shown by the marked bone destruction. The cavernous sinus thrombophlebitis is a progression from the pericarotid plexus phlebitis. The infection of the Gasserian ganglia, with no direct extension seen, is probably phlebitic in origin.

Case No. 6746.—White female, age 24 years. Admitted January 2, 1937, died January 13, 1937. On entrance the patient, who was two months pregnant, had left otitis media of six day's duration. Patient acutely ill. First degree nystagmus to left and vertical nystagmus on looking upwards. Purulent discharge from central perforation on left; mastoid, tender. Hearing was down, but pres-

ent, in the left ear, and the labyrinth reacted to caloric stimulation. Temperature up to 101 degrees. X-ray showed hazy mastoid. On January 8, 1937, about twelve days after onset, left simple mastoidectomy was performed. Mastoid well pneumatized, pus present throughout. A tract of cells behind the labyrinth, extending inward was curetted for about 1 cm. Middle fossa and lateral sinus dura appeared normal.

Culture from mastoid and blood showed hemolytic streptococcus.

Developed right otitis media on January 8. Vomited postoperatively. Question of vomiting of pregnancy. Temperature 39.2 degrees.

On January 13 the left pupil was widely dilated and fixed to light. Patient semi-comatose. Ptosis left eye. Spontaneously aborted a two months' fetus. Later the right pupil dilated and would not react. Died the same day.

Autopsy: Purulent meningitis and purulent sphenoiditis.

Microscopic Observations: Unilateral apicitis.

Left Ear: There is an acute purulent otitis media and mastoiditis. A huge cell extends into the postero-superior angle of the pyramid and occupies much of the superior aspect of the apex (Fig. 2). This cell is full of pus. Its mucosa is in direct contact with neighboring marrow, which shows an early acute osteomyelitis. In one section a blood vessel, whose walls show inflammation, connects mucosa and marrow. The infected marrow is in contact with the dura under the Gasserian ganglion through a dehiscence (see X, Fig. 2). The dura is infiltrated and there is pus in the Gasserian ganglion.

Badly infected hypotympanic cells also extend into the apex. In the midpoint of the latter the suppurative process is most severe. Here is a huge coalescent abscess with no normal structure remaining, and an extreme degree of bone destruction. It seems to have occurred mainly in air containing cells (Fig. 3), although there is also an acute osteomyelitis in marrow containing cells at the extreme tip. A fistula connects this coalescent area with the posterior fossa.

The round window membrane is discolored and there is a purulent labyrinthitis, both in vestibule and cochlea. The preponderance of pus is in the perilymphatic space. Pus is seen, however, in the ductus endolymphaticus.

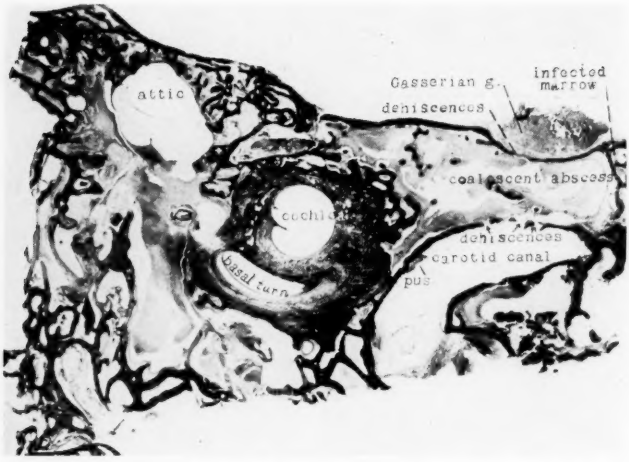

Fig. 1. No. 6379 Right. Age 6 years. White male. Vertical longitudinal section. Coalescent osteitis and acute osteomyelitis. Dehiscence beneath Gasserian ganglion with intact dura. Pus present within ganglion, however. Multiple dehiscences into and pus within carotid canal.

Right Ear: This side shows pneumatization exactly symmetrical with that on the left side. Though there is acute otitis, mastoditis, and pus in the many posterior perilabyrinthine cells, there is, however, no pus in the large cell which extends into the apex, as may be seen in Fig. 4. There is some pus in a hypotympanic cell which is just anterior to the jugular bulb. There is also pus in the cranial ostia of the aqueductus cochleæ and in the internal auditory meatus, retrograde from the meningitis.

Comment: On the left side a severe suppurative apicitis, occurring in both air and marrow containing cells. There are multiple portals for the infection of the meninges.

The bones, with their symmetrical pneumatization, graphically reveal the progress of the disease. On the right a more acute ear infection is present in the posterior perilabyrinthine cells, but has not as yet extended to the apex. Had the patient lived longer progression to the apex would have undoubtedly occurred on this side, with the end result as seen on the left.

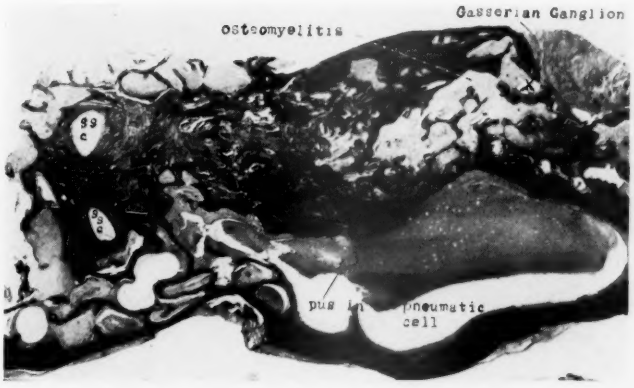

Fig. 2. No. 6746 Left. Age 24 years. White female. Horizontal section. Superior level. Huge pus-filled pneumatic cell. Osteomyelitis in diploetic cells beneath Gasserian ganglion which is exposed to this infected area. Cell tract forward, medial to arcuate eminence. *X* marks dehiscence under Gasserian ganglion.

CASE NO. 6358 (courtesy of Dr Arbuckle). White male, age 16 years. Admitted January 7, 1936, died January 10, 1936. The patient, a resident of Indiana, developed bilateral otitis media about three weeks before admission to Barnes Hospital. Right sixth nerve weakness was noted ten days before admission, and a right simple mastoidectomy was done in Evansville, Indiana, five days before admission. Following this, patient improved for three days, but then developed headache and vomiting. Spinal fluid now was cloudy. On admission to Barnes Hospital he had stiff neck, bilateral Kernig and a right sixth nerve weakness. Dried secretion in left canal.

On January 7 a laminectomy was done for drainage. Spinal fluid contained 4300 cells, mostly polys. Culture showed pneumococcus type I. Blood culture positive for same organism. On January 9, given 20,000 units each of type I and II pneumococcus serum. Despite previous negative tests for sensitivity when about 80 per cent of serum was given, he had convulsions, after which he remained comatose. The next day serum repeated. When about 2.5 cc. had been given, breathing ceased, and despite treatment, including respirator, he died fifty minutes later.

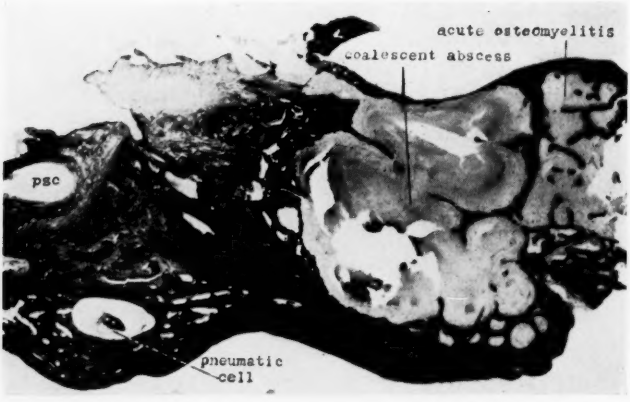

Fig. 3. No. 6746 Left. Age 24 years. White female. Infracochlear level. Coalescent abscess with marked bone absorption. Acute osteomyelitis of marrow cells at tip.

Mastoid x-ray showed bilateral mastoiditis (operative defect right). Submento-vertical and Towne positions of the petrous pyramid showed some increase in density of the cells over the right petrous pyramid.

Autopsy: Purulent meningitis. The portal of entry seemed to be about the right internal auditory meatus.

Microscopic Observations: Bilateral apicitis.

Right Ear: There is marked perilabyrinthine pneumatization. Cell tracts lead to the apex above, medial and lateral to the arcuate eminence. Chains of cells also lead to the apex above the internal auditory meatus and by way of the subarcuate fossa. Hypotympanic cells can be traced into the apex anterior to the ascending part of the carotid artery. The periantral and perilabyrinthine cells, as well as the middle ear, are lined by greatly thickened, heavily infiltrated mucosa and there is some purulent exudate in their lumina.

Anterior to the superior semicircular canal the cortex is eroded, and there is a marked dural reaction with great thickening of the dura and new bone formation.

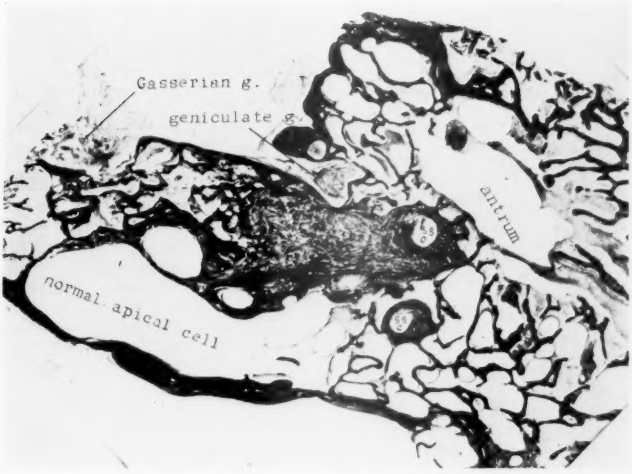

Fig. 4. No. 6746 Right. Age 24 years. White female. Horizontal section. Superior level. Note normal apical cell in the presence of involved perilabyrinthine cells.

The apex is occupied by a huge abscess which seems to have arisen by a coalescence of cells (Fig. 5). The walls of this abscess consist of granulation tissue. There is great bone destruction and much new bone formation. This infected apex is in contact with the dura under the Gasserian ganglion, but the dura is not penetrated and the ganglion is normal. In the region of the sixth nerve the cortex is eroded, the dura infiltrated throughout. Likewise more posteriorly there is a large pathological dehiscence of the posterior fossa and here the purulent contents of the apical abscess are spilling into the dura through a chronic fistulous tract. This is most probably the source of infection of the meninges. The severe inflammatory process of the apex has also eroded into the internal auditory meatus, where the dura is infiltrated, thickened, and there is much new bone formation.

There is a subperiosteal abscess in the external auditory canal.

Left Ear: In this ear the pneumatization is symmetrical with that on the right side. The cell tracts forward to the apex are identical (Fig. 6). The pathological process seems somewhat more

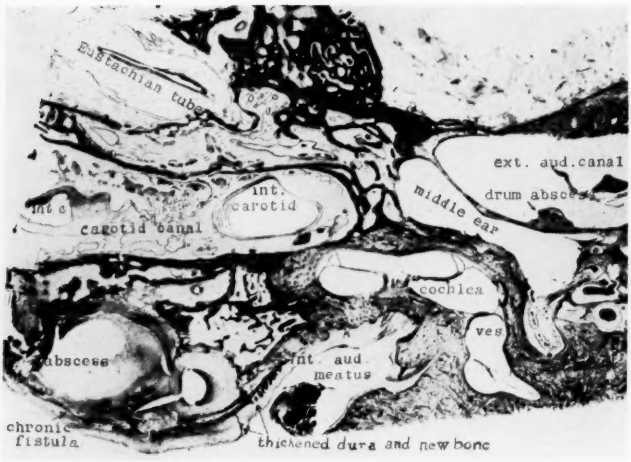

Fig. 5. No. 6358 Right. Age 16 years. White male. Horizontal section. Inferior level. Coalescent apical abscess spilling contents into dura of posterior fossa through chronic fistulous tract. Note thickened dura and new-formed bone in internal auditory meatus.

low-grade. It is characterized in the periantral, peritubal and perilabyrinthine cells by marked swelling of the mucosa, which is infiltrated mainly with lymphocytes and plasma cells. There is a minimum of purulent exudate. Likewise, the process in the apex is similar to that in the right ear, but instead of an abscess, the superior two-thirds of the apex is occupied by an inflammatory cyst, its walls consisting of heavily infiltrated granulation tissue, its contents, a small amount of serum with a few cells. There is much bone destruction and new bone formation, as shown in Figs. 6 and 7.

A thrombosed vein is seen in the dura close to the Gasserian ganglion. There are several dehiscences under the ganglion. At one of these the dura is involved throughout by inflammatory cells and here there is pus in the Gasserian sheath. This could well have been an additional source of infection of the meninges (Fig. 8).

There is pus in the facial canal, and a subperiosteal abscess in the carotid canal.

Comment: A low-grade type of process of about one month's duration, characterized bilaterally by a marked granulation tissue

Fig. 6. No. 6358 Left. Age 16 years. White male. Horizontal section. Superior level. Cell tracts leading forward to apex around both sides of arcuate eminence, i. e. laterally from attic and also medially. Subacute inflammation with empyemic membrane in pneumatic cells and consequent obliteration of their lumina. Granulations and young bone in apex. Note bloodvessel *(bv.)* penetrating tegmen.

response and a tremendous capacity for bone destruction. Multiple dehiscences on both sides, several of which might have been the portal of entry to the meninges. Complete involvement of the dura in the region of Dorello's canal on the right, and clinically a right sixth nerve weakness.

Case No. 3951.—White female, age 5 years. Admitted November 20, 1929, died November 21, 1929. Nine weeks before admission developed head cold and earache. Left ear ruptured spontaneously after three days. Drainage continued and patient seemed to be in good condition until one week before admission, when patient seemed drowsy. Complained of headache and had irregular fever. On day of admission headache more severe and patient became semi-stuporous. Vomited once. On admission temperature 38.6 degrees. Breathing irregular. Profuse discharge from the left ear.

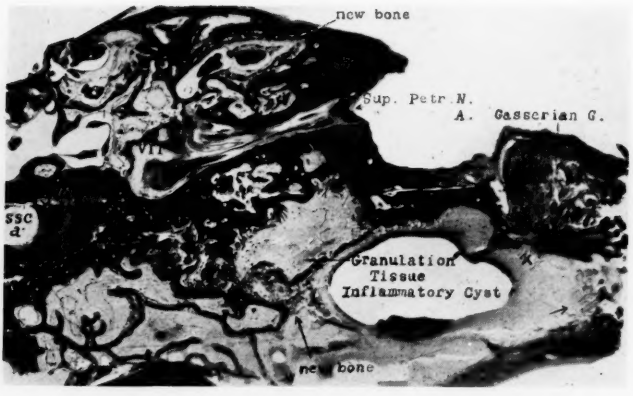

Fig. 7. No. 6358 Left. Age 16 years. White male. View 20 sections below Fig. 6. Entire apex replaced by infected granulations with cystic center. New bone formation. X marks dehiscence and complete involvement of dura under Gasserian ganglion.

Typical signs of meningitis. White blood count, 20,000. Spinal fluid turbid, cells mainly polymorphonuclear leucocytes. Spinal fluid culture showed streptococci. Patient died next day.

Autopsy: Purulent meningitis. Thrombosis of the sinus at the junction of left superior petrosal and left lateral sinus. Also thrombosis of left cavernous sinus, and a small thrombus in the sagittal sinus.

Microscopic Observations: Bilateral apicitis.

Left Ear: There is a severe chronic otitis media and mastoiditis. The mucous membrane of the tympanic cavity, eustachian tube and mastoid air cells is greatly thickened by fibrous tissue and inflammatory cells. In the middle ear and tube is polypoid tissue. The apex, except for peritubal cells which curve around the ascending portion of the carotid, is diploetic. These peritubal cells have become lined with columnar, ciliated epithelium and are in contact with the marrow of the apex. The apical marrow at a superior level shows a severe degree of fibrosis (Fig. 9) and invasion with lymphocytes, plasma cells and histiocytes. However, at the level of the

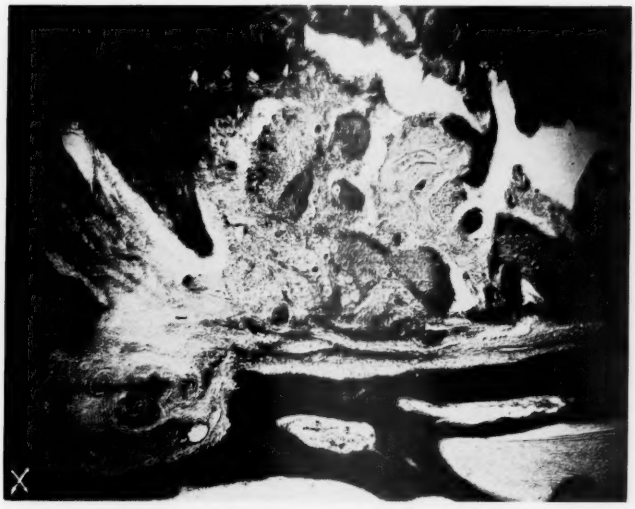

Fig. 8. No. 6358 Left. Age 16 years. White male. Detail of fistulous tract from apex into Gasserian ganglion. This view is 70 sections below that of Fig. 7.

promontory there has been much bone destruction, so that here the apex has been replaced by a coalescent mass of heavily infected granulation tissue, in which are a few small abscesses and some new-formed bone. From this there has been developed a chronic fistulous tract which extends through the dura of the posterior fossa, as shown in Fig. 10. Inferiorly from this area of greatest involvement there is what appears to be a severely infected fistulous tract which extends into the region of Rosenmuller's fossa. At its highest level it contains some pus, and throughout its course the bone is destroyed. There are several dehiscences into the carotid canal, and the periosteum of the canal is thickened. Veins of the pericarotid plexus are filled by infected thrombi. Several infected thrombi are seen in veins of the Gasserian ganglion, and the latter is infiltrated in areas by pus.

Right Ear: There is a slight degree of chronic otitis media and mastoiditis. The eustachian tube and peritubal cells show the more

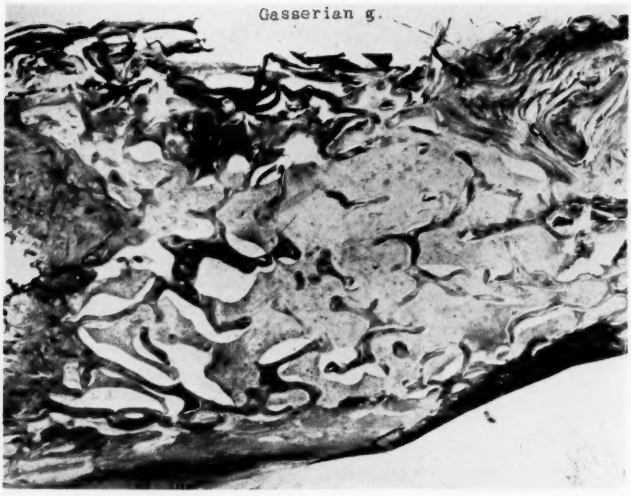

Fig. 9. No. 3951 Left. Age 5 years. White female. Horizontal section. Superior level. Showing apical region only, in which is marked fibrosis of diploic structure. Thickening of dura of Gasserian sheath.

marked chronic infection. The tubal mucosa is polypoid. There is partial pneumatization of the apex by way of the peritubal cells. These apical cells show a very slight early infection with hyperemia and thickening of the mucosa and a serous exudate in which are a few pus cells. The marrow of the apex is relatively normal. A subperiosteal abscess is present in the carotid canal. There is an infected thrombus in the superior petrosal sinus, and the Gasserian ganglion is infiltrated by a huge amount of pus. This ear is also interesting, for in a part of its descending route the facial nerve is replaced by bone.

Comment: Mainly an osteomyelitis on one side and a slight pneumatic cell infection on the other. There is opportunity here for infection of the meninges by direct extension and through venous channels, though the former is the predominant pathway. The Gasserian ganglia are probably infected by the latter route. It would seem that this took place from the apex on the left but by retrograde thrombosis on the right.

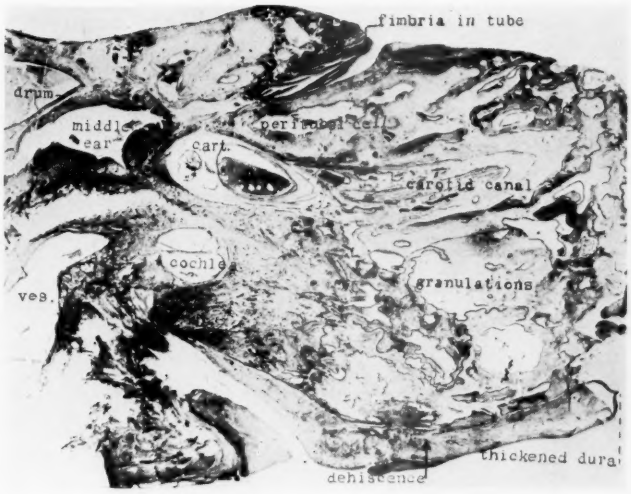

Fig. 10. No. 3951 Left. Age 5 years. White female. Inferior level. Note chronic fistulous tract at point of dehiscence. Entire apex is a coalescent mass of heavily infected granulation tissue, the pathological condition extending, apparently, from peritubal cells.

CASE No. 5680 (courtesy of Dr. Lyman). White male, age 30 years. Admitted March 8, 1934, died April 9, 1934. Entered Barnes Hospital with discharging left ear of five week's duration. X-ray showed considerable destruction of cellular structure on the left. Examination showed signs of mastoiditis and a left simple mastoidectomy was done on March 9, 1934. It was an extensively pneumatized mastoid process, the cells containing greyish membrane. Dura and sinus not exposed. Postoperative course very uneventful, and patient discharged on March 17, 1934.

He returned April 6, 1934. Had done well until ten days previously, when he had developed occipital headache radiating to mastoid, and for past forty-eight hours he had been vomiting and had vertigo. Fundi normal. Spontaneous nystagmus on looking to right and left. No change in nystagmus on douching. Hearing in left ear was present. No meningeal signs. It was felt that he had a serous labyrinthitis. Spinal fluid revealed 200 lymphocytes per cu. mm. On April 6 an x-ray in the Towne position showed an in-

crease in density throughout the left petrous pyramid with irregular loss of continuity of the superior cortical outline of the pyramid. The x-ray diagnosis was questionable osteomyelitis of the left petrous pyramid. On April 6 he was reoperated. Behind and below the labyrinth soft bone was found, and about five-eighths inch internal to the floor of the antrum a large cell containing necrotic material was found. In the depths of this cell, dura of the posterior fossa or jugular bulb seemed to be visible.

On April 7 there was no fever, but the patient complained of diplopia, with headache, nausea, vomiting, drowsiness. Spontaneous nystagmus to right and left. Condition remained poor. On April 9 had ankleclonus, questionable right Babinski, was restless and involuntary; 204 cells, all lymphocytes, found in spinal fluid. Patient died one hour later the same day.

The clinical diagnosis was osteomyelitis of the left petrous pyramid, acute brain abscess, localized meningitis? and labyrinthitis? The organism from the original myringotomy was a pneumococcus, type unknown.

Autopsy: Revealed an abscess in the left lobe of the cerebellum.

Microscopic Observations: Unilateral apicitis.

Left Ear: There are numerous posterior perilabyrinthine cells showing a chronic process with thickened, infiltrated mucosa with a minimum of purulent exudate in their lumina. Tympanic cavity likewise shows a chronic process with thickened, infiltrated polypoid mucosa, especially over promontory. Same chronic process in epitympanic cells. Cells medial and lateral to superior canal extend forward to the apex. A few cells of the apex can be traced from peritubal cells. Just anterior to superior canal is a huge coalescent process with massive granulations, bone destruction, much new bone formation (Fig. 11). The posterior-superior coalescence is very large, extending below the level of cochlea. The operative fistulous tract has extended behind the labyrinthine capsule and connects with this granulating area. This posterior-superior angle coalescence involves the dura at a superior level and is undoubtedly the source of cerebellar abscess. The apex contains few pneumatic cells with thickened, infiltrated mucosa and new bone formation. The rest of the apex shows an osteomyelitis with the fatty marrow revealing fibrosis, lymphocytic and plasma cell infiltration. This inflammatory process has caused much bone absorption and there is an extreme degree of new bone formation. Only a few fat cells remain, especially at the very apex and at the inferior levels. This infected

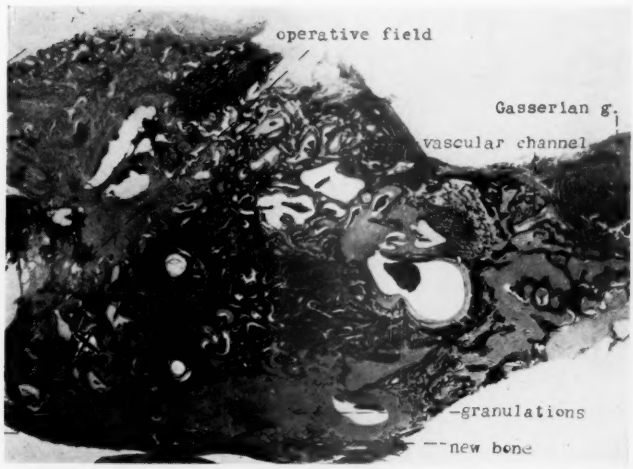

Fig. 11. No. 5680 Left. Age 30 years. White male. Horizontal section. Superior level. Operative fistulous tract leading into granulating area at X. This continues forward to extreme apex. Dura involved, however, from posterior-superior angle coalescence and not from Gasserian ganglion region.

marrow is in contact with the dura under the Gasserian ganglion by numerous small dehiscences, but the dura is not penetrated and the Gasserian ganglion is normal.

Right Ear: Not infected. Markedly pneumatized by superior route, almost complete apex occupied by air cells. Only most anterior tip under Gasserian ganglion contains fatty marrow.

Comment: A chronic process characterized by granulations, severe bone destruction and production. Apex shows reactive, proliferative osteitis and osteomyelitis. Though a marked apicitis is present the important lesion is in base of petrosa in the posterior superior aspect.

CASE No. 6090.—White female, aged 9 years. Admitted January 23, 1935, died April 4, 1935. Patient admitted to Children's Hospital with bronchiectasis, sinusitis and chronic tonsillitis and adenoids. On January 30, 1935, bilateral antral windows and tonsillectomy and adenoidectomy were done. Discharged to convalescent

farm in good condition. On February 13, complained of pain in right ear and on February 16, was returned to Children's Hospital with bilateral otitis media. Both ears drained well. On March 2, again returned to convalescent farm, but returned to hospital on March 6 with left mastoid tenderness. X-ray report of bilateral mastoiditis. Left mastoidectomy contemplated, but deferred because of otherwise unexplained rise of temperature to 38.5. Mastoid x-rays remained the same. Ears seemed to improve, temperature decreased, tenderness diminished and child again went to convalescent home on March 23. Re-entered Children's Hospital on April 1 because of temperature up to 39 degrees, severe frontoparietal headache and vomiting. The ears were not discharging, but the drums were not normal and there was a granuloma on the right coming from the region of Shrapnell's membrane. This was removed. It was felt now that the ears might be responsible for all the trouble.

Frequent fundus examinations revealed normal findings.

Patient was running low-grade temperature and seemed to be improving, but was found dead in bed on April 4, 1935.

Autopsy: Revealed a left cerebellar abscess. Culture of pus from abscess revealed hemolytic streptococci.

Microscopic Observations: Unilateral apicitis.

Left Ear: The periantral, posterior superior perilabyrinthine cells, zygomatic cells and middle ear reveal evidence of long-standing inflammation. There is marked fibrosis of the mucosa and new bone formation. There is also an acute infection superimposed, with purulent cellular exudate and pus in Prussak's space. Cells extend over and medial to the arcuate eminence, and in the posterior superior angle anterior to the arcuate eminence is a huge abscess with much bone destruction and new bone formation. This has eroded the cortex of the posterior fossa and the dura here is a mass of pus. This intradural abscess is one point of invasion of the cerebellum. This abscess also has dissected forward in the posterior fossa to the internal auditory meatus, and here the dura is greatly thickened and there is new bone formation, showing a process of considerable duration. Moreover there is erosion of the temporal bone by this process. A patent subarcuate fossa is also a route of invasion to the intracranial contents. The dura here is abscessed, bone is undergoing absorption and there is great osteoclastic activity. (Fig. 12.)

No communication is seen with the diploetic apex, but a chronic osteomyelitis is present. The apical marrow shows considerable

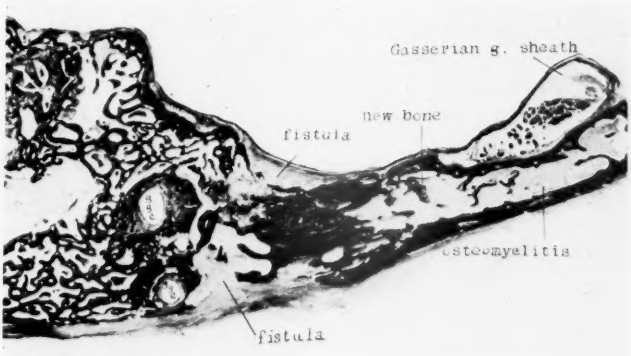

Fig. 12. No. 6090 Left. Age 9 years. White female. Horizontal section. Superior level. Note distended Gasserian sheath enveloped by intact dura. Fistulous tract into posterior fossa is seen in the area of subarcuate fossa. This is the site of invasion of the cerebellum.

fibrosis, bone absorption and much new bone formation. There are likewise several small abscesses present here. The infected marrow is in contact with the dura under the Gasserian ganglion, but no invasion has occurred, the Gasserian sheath being intact. (Fig. 12.)

Right Ear: Shows attic perforation, early cholesteatoma formation, chronic mastoiditis and otitis media. No petrositis or apicitis. The marrow of the apex is relatively normal.

Comment: A long-standing infection of the petrous pyramid in which, although the apex is involved, the more severe and fatal lesion is located in the posterior superior angle of the pyramid. The lesion in the apex is mainly a reactive, reparative one with small, isolated areas of suppuration.

CASE NO. 5563.—White male, aged 7 years. Admitted December 19, 1933, died January 15, 1934. Eight days before admission patient developed fever and had pain in left ear. Myringotomy the next day with little improvement. Severe frontal headache and fever. Vomited several times. Stiff neck for three days before admission. History of frequent trouble with ears since age of two. One month ago had earache that subsided in twelve hours. On ad-

mission patient acutely ill with draining left ear and neck slightly rigid. Meningitis considered, but not thought likely. A consultant noted sagging of the canal wall and felt that the process was of longer duration than the history indicated. Improvement on conservative measures and temperature normal on January 1. On January 2, septic temperature from 38 to 40.5 degrees developed. On January 5, developed measles. On January 9, seemed better but on January 10, he complained of headache, vomiting and meningeal signs appeared. Blood culture previously sterile now showed hemolytic streptococcus. Lumbar puncture showed 400 cells and streptococci on smear. Laminectomy and forced drainage was done. Patient developed pneumonia and died on January 15, 1934.

Mastoid x-rays showed cloudiness on the left with some dissolution of structure. Films of petrous pyramid not taken.

Autopsy: Revealed a purulent meningitis and a broncho-pneumonia. Intracranial venous sinuses not thrombosed.

Microscopic Observations: Bilateral apicitis.

Left Ear: Middle ear cavity, antrum and periantral cells show cystic scar tissue, evidence of an old inflammation. Superimposed on this is an acute purulent reaction. Epitympanic cells extend forward to the apex where there is one cell containing pus (Fig. 13). The dura of the subarcuate fossa is in contact with the infected petrosal cells, but is not involved. The marrow of the apex is in contact with the infected apical cell, and reveals a subacute osteomyelitis with hyperemia, diffuse cellular infiltration including that of numerous polymorphonuclear leucocytes. This infected marrow is in contact with the dura under the Gasserian ganglion, but both the dura and the Gasserian ganglion appear intact, though the former reveals a reactive thickening. At progressively more inferior levels the infection becomes more severe until there is a definite suppurative reaction with bone destruction and abscess formation, and at low levels a large abscess almost surrounds the carotid. This reaction mainly involves air cells. Here a second and more severe route of infection is seen; infected hypotympanic cells can be traced and seen in contact with the apical marrow.

Right Ear: An acute reaction is present in middle ear and mastoid cells, but not so marked as on the left. There are fewer posterior perilabyrinthine cells, and no superior cell tracts can be traced to the apex. Peritubal cells contain pus and at the level of the foot-plate of stapes such a cell is continuous with a large apical cell which almost surrounds the ascending portion of the internal

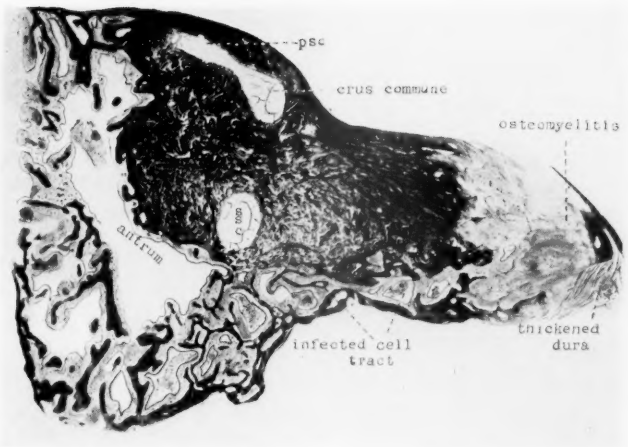

Fig. 13. No. 5563 Left. Age 7 years. White male. Horizontal section. Superior level showing infected cell tract leading to apex which is the seat of a subacute osteomyelitis.

carotid. There is a thin layer of pus in this cell, and its mucosa is in contact with marrow, but the latter shows no reaction other than hyperplasia.

There is hemorrhage in the pericarotid sheath and a subperiosteal hemorrhage in the carotid canal. The round window membrane is infiltrated and there is pus in the basal coil.

Comment: On the left is a mixed type of infection in a mixed type of apex, but the infection in the air cells predominates. Infection has reached the apex both by the superior petrosal route and the peritubal route. The latter here is of most importance, as evidenced by the greater pathological process at the inferior level. Infection to, but not through, the dura. No direct route to meninges seen; probably a hematogenous meningitis without thrombophlebitis (clinically a positive blood culture). In the right ear a terminal, slight infection in the apex.

Case No. 4114.—White female, age 4 years. Admitted May 31, 1930; died June 4, 1930. Spontaneous discharge from left ear

three weeks before admission. High fever for one week, severe headache, pain in back of neck. Progressive listlessness and drowsiness. Temperature on admission 37 degrees. Signs of mastoiditis and suggestive meningeal signs. Spinal fluid cloudy, 1000 cells, mostly polys. No organisms seen. Simple mastoidectomy on June 1. Pus and granulations found and dura of middle fossa exposed. Granulations seen on dura which appeared tense. On June 2 temperature to 40 degrees, 5000 cells in spinal fluid, hemolytic streptococci on culture. Cisternal drainage done and tube sewed intradurally. Temperature to 40.8 degrees, generalized convulsions and child died on June 4, 1930.

Autopsy: Revealed purulent meningitis.

Microscopic Observations: Unilateral apicitis.

Left Ear: The periantral cells reveal a subacute mastoiditis with infiltrated empyemic membrane, and only a small amount of pus. Cells extend into the petrosa at the postero-superior angle, and through the subarcuate fossa. The apex is wholly diploetic and the marrow cells appear directly continuous with pneumatic cells over the internal auditory meatus. These pneumatic petrosal cells show the most acute reaction with many polymorphonuclear leucocytes and there is an abscess in the subarcuate fossa where there is new bone formation. From these cells are several dehiscences through the cortex with involvement of the overlying dura.

The apex at the superior level reveals a severe acute suppurative osteomyelitis with bone destruction. There is a large pathological dehiscence here, with the pus breaking under the dura and also causing an intradural abscess (Fig 14). This is certainly the origin of the intradural abscess, despite the multiple dehiscences, as here the infection is most acute. This extradural and intradural abscess has dissected the layers of the dura and can be found as far posteriorly as the tegmen antri, at which point the process is more reactive, with lymphocytes and plasma cells. The dura throughout is greatly thickened, the cortex is being eroded in numerous places from without inwards, and there is new bone formation. In the region of the internal auditory meatus the intradural abscess has involved all layers of the dura. This is at least one of the portals of entry to the meninges. There is a thrombophlebitis of the superior petrosal sinus and of numerous small, intradural veins.

Below the suppurative osteomyelitis of the superior apex the marrow reveals a reactive, proliferative fibrotic reaction with lymphocytic and plasma cell infiltration.

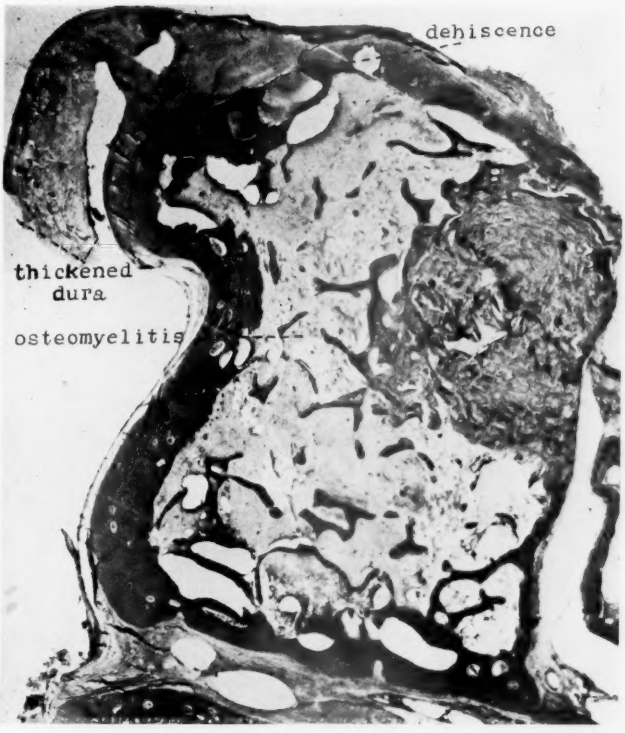

Fig. 14. No. 4114 Left. Age 4 years. White female. Vertical section. Apical region. Pathological dehiscence producing intradural abscess. Note severe acute suppurative osteomyelitis.

Comment: Apical marrow infected by direct continuity from petrosal, nonapical cells. This is shown by the more severe reaction at superior levels, with a purely proliferative reaction at lower levels, which is an obvious attempt at limitation of the osteomyelitis. The apical infection is more acute and severe than that in the mastoid. It is, however, of considerable duration, as evidenced by the marked dural reaction with bone formation, and the dissecting intradural abscess.

CASE No. 31 (Courtesy of Dr. Alden).—White male, aged 4 years. Admitted to St. Louis County Hospital because of fever,

pain, discharge from left ear. Pain in left ear started two days before, with spontaneous rupture and discharge in six hours. Temperature on admission 105 degrees, child septic. W. B. C. 14,000 with 27 stabs. X-ray showed haziness of left mastoid. Blood culture negative. Two days after admission chill and stabs rose to 42. Slight tenderness over mastoid. Photophobia and slight nuchal rigidity. Simple mastoidectomy done. All cells filled with thin pus. Sinus opened exposing a mural thrombus. Free bleeding from above, not from below. Due to child's poor condition jugular not tied. Next day W. B. C. 8300, stabs 31. Temperature high and child irrational. Marked edema of left side of face, which involved eye, cheek and neck. Neck rigid, child died early next morning.

Autopsy: Revealed purulent meningitis. Cultures from operation and autopsy revealed hemolytic streptococci.

Microscopic Observations: Unilateral apicitis.

Left Ear: Although there are some cells postero-superiorly about the labyrinth, the apex is entirely diploetic. There is an acute purulent reaction in the middle ear, periantral and perilabyrinthine cells. Infected hyptoympanic cells are continuous with infected marrow cells which run forward to the apex under the labyrinth. This is likely one source of infection of the apex. There are multiple submucosal tubal abscesses, and a subperiosteal abscess medial to the tensor tympani. Pus in the eustachian tube is entering a dehiscence in the carotid canal and there is a subperiosteal abscess. Here is marked periosteal thickening and bone formation, possible evidence of previous infection of the carotid canal. At the level of the isthmus (Fig. 15) pus from a submucosal tubal abscess is continuous with the marrow of the apex, and in the apex is an acute, diffuse purulent osteomyelitis with beginning bone absorption.

A huge abscess is present in the carotid sheath. This seems to have its origin in a phlebitis of the pericarotid venous plexus, as the remnants of vein walls may be seen in the midst of the abscess, and a few thrombi are also seen. This abscess is also in contact with the apical marrow through a dehiscence.

The pus in the marrow has caused a dehiscence of the inferior surface of the bone, and here is a subperiosteal (retropharyngeal) abscess which has stripped most of the periosteum from the bone. In the retropharyngeal tissue are multiple miliary abscesses. These apparently owe their origin to a retrograde thrombosis of the veins of these tissues. Occasional thrombi are seen. There are infected thrombi in intradural veins in the region of the Gasserian ganglion

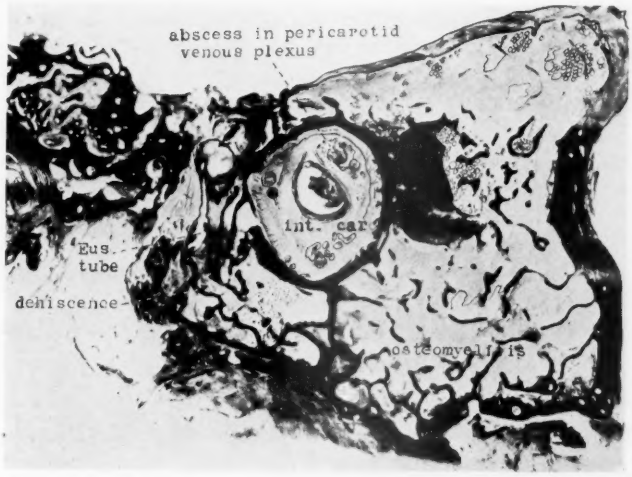

Fig. 15. No. 31 Left. Age 4 years. White male. Vertical section. Region of tubal isthmus. Note pus from submucosal tubal abscess entering apical marrow. Another abscess is present within the carotid canal arising from pericarotid venous plexus.

and an intradural abscess, but the ganglion is not involved and no direct extension through the dura is seen. Also noted is an acute osteomyelitis of the lesser wing of the sphenoid.

There is a fistula through the floor of the hypotympanum into the jugular fossa, with a perijugular bulb abscess, a jugular vein phlebitis and thrombosis.

In the external auditory canal is a subcutaneous abscess. Blood is present in the internal auditory meatus and there is hemorrhage in the perilymphatic space of the posterior canal.

Comment: This case probably is the type of osteomyelitic phlegmon described by Eagleton. There is an associated multiple phlebitis, and the fatal infection is a fulminating phlebitic meningitis. Thus infected veins are seen in the dura; a similar process has likely occurred in the pia arachnoid. The cavernous sinus was not removed, but there was undoubtedly a phlebitis here, as evidenced clinically by the edema of the face and pathologically by the multiple retropharyngeal abscesses having their origin in small veins (retrograde

from the cavernous sinus). The case is likewise interesting as showing two methods of formation of retropharyngeal abscesses from suppurations of the petrous apex, retrograde thrombosis and direct extension through the inferior cortex.

CASE No. 5518. Negro male, age 7 years. Admitted November 2, 1933; died December 3, 1933. Patient had a left simple mastoidectomy in June, for a discharging ear of four months' duration. At this operation necrotic cells were found throughout, but no free pus. Following this operation the discharge continued and he entered Children's Hospital on November 2, 1933, with an acute exacerbation and postauricular swelling. A second simple mastoidectomy was done on November 4, 1933. A subperiosteal abscess was incised, granulations filled the mastoid cavity. Antrum enlarged, few zygomatic cells removed, dura not affected. Culture revealed streptococcus viridans.

Patient progressed satisfactorily but profuse discharge continued. Temperature on November 14 suddenly rose to 104 degrees. Neck stiff. Lumbar puncture revealed turbid fluid, 4500 W.B.C. per cm. Smear revealed 80 per cent polys and occasional groups of cocci. On November 15 lumbar laminectomy was done and the mastoid reoperated. Granulations were removed from the antrum, and the tegmen antri and tympani appeared soft and necrotic and were removed. The dura here looked unhealthy and was bulging. Course rather stationary but temperature remained high. On November 23 the laminectomy had ceased drainage and cisternal drainage by catheter was done. Despite this, course was downhill and the patient died on December 3, 1933.

No x-rays of petrous pyramids were taken.

Autopsy: Revealed purulent meningitis.

Microscopic Observations: Unilateral apicitis.

Left Ear: There is a large number of cells in the area of Trautman's triangle and many posterior perilabyrinthine cells are present. A tract of cells leads forward through the subarcuate fossa but does not reach the apex. These cells and the antrum show a chronic process with thickened, infiltrated mucosa and a minimum of acute reaction. The dura in the subarcuate fossa is not involved. There is new bone formation in the attic region.

There is pneumatization of the infero-lateral cells of the apex, the cells of communication extending from the hypotympanic area

to the apex between cochlea and carotid. These cells reveal a chronic process similar to that of the mastoid, and there are many cholesterin crystals in the thickened mucosa. At an inferior level a perpendicular plate of bone separates the lateral pneumatic cells from the medial diploetic cells. The neighboring diploetic cells show a reactive fibrotic process with cellular infiltration. Likewise at a higher level the infected pneumatic cell mucosa is in direct contact with marrow, and here again the marrow shows a proliferative type of reaction. At the superior level the marrow is quite normal.

There is pus in the internal auditory meatus.

Right Ear: Not infected. Anatomically not symmetrical with left ear, as superior petrosal cells have extended forward to pneumatize the entire apex. There is pus at the ostia of the aqueductus cochlea and in the jugular ganglion, retrograde from the meningitis.

Comment: A chronic infection, with the same type of reaction in mastoid cells and in the cells of the partially pneumatized apex. Marrow of the apex also reveals a low-grade inflammatory process where it is close to and in contact with the infected pneumatic cells. The route of extension to the meninges is not seen. From a clinical standpoint suppuration in the petrous pyramid should have been strongly suggested by the continuance of discharge when the two revisions of the simple mastoidectomy revealed very insignificant pathological processes.

CASE No. 6335. White male, age 7 years. Admitted December 16, 1935; died December 16, 1935. One week prior to admission developed severe cold and left earache. Ear discharged spontaneously. Four days prior to admission aroused with difficulty and hardly ate or drank. One day before admission had severe chill, complained of headache and vomited several times, and a few hours before admission had a severe chill and possibly a convulsion. Became delirious and rigid. On examination was comatose. Breathing Cheyne-Stokes. Discs choked. Thick pus came from left ear. Neck rigid, Kernig and Brudzinski positive. Temperature 40.8 degrees. Expired six hours after admission during performance of lumbar puncture. Postmortem blood and spinal fluid culture positive for streptococcus hemolyticus. Spinal fluid cloudy, 800 cells, 4 + Pandy.

A sister developed otitic meningitis at the same time and died after five weeks.

Autopsy: Revealed a purulent meningitis.

Microscopic Observations: Bilateral apicitis.

Right Ear: This ear reveals an acute process, with purulent exudate, superimposed upon a subacute infection, as shown by marked swelling of the mucosa which is infiltrated mainly by lymphocytes and plasma cells. There are cholesterin crystals in periantral cell mucosa. The same pathological process is present in antrum, periantral cells, tympanic cavity and peritubal cells. There is no posterior perilabyrinthine pneumatization but the peritubal cells are continuous with pneumatic cells which occupy the inferior aspect of the petrous apex. These apical cells reveal a subacute inflammatory reaction without purulent exudate. The mucosa of these cells is directly continuous with the marrow cells of the apex, and these marrow cells reveal a reactive process in the form of fibrosis, hyperemia, lymphocytic and histiocytic invasion. The marrow of the superior aspect of the apex, farther removed from this infective focus, is hyperplastic.

The superior cortex is intact. An infected thrombus is seen in the dura of the middle fossa at the tip and there is much pus in the Gasserian ganglion.

Left Ear: This was sectioned in the vertical longitudinal plane. It is anatomically symmetrical with the right one. The inferior aspect of the apex is pneumatized, and the cells can be traced to the peritubal cells. The middle ear and mastoid infection is more acute and more severe than on the opposite side, with more frank pus and less empyemic membrane.

The apex reveals a severe coalescent osteitis at the inferior aspect and an acute osteomyelitis with abscess formation in the diploetic cells. Bone absorption and cholesterin crystals occur in some of the abscesses. The more severe infection is inferiorly, dependent on the point of invasion. There is infected marrow in contact with the dura under the Gasserian ganglion, but the dura is not involved. There are multiple partially organized thrombi in the veins of dura of the middle fossa over the extreme apex, and one thrombosed vein is seen in the midst of the Gasserian ganglion. The walls of this vein are infiltrated with inflammatory cells. There is a large abscess of the Gasserian ganglion (Fig. 16) and pus and fibrin on the surface of the dura.

Comment: A bilateral osteitis and osteomyelitis in mixed types of apices. The process is more severe and destructive on the left. Nevertheless there is evidence of a thrombophlebitis on both sides, and this is most likely the source of meningeal and Gasserian ganglion

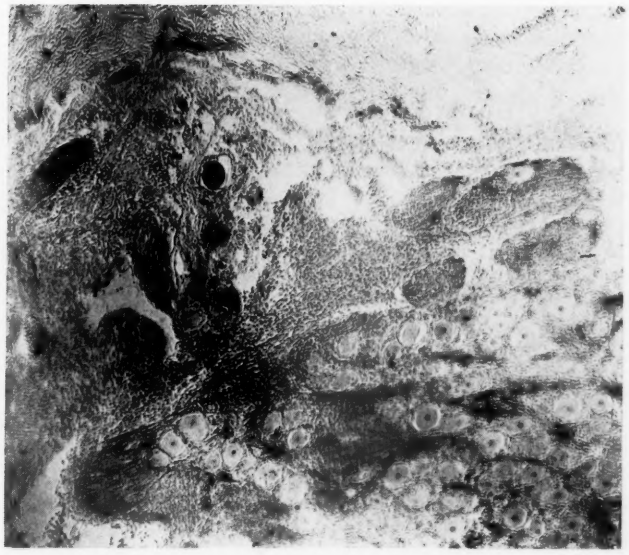

Fig. 16. No. 6335 Left. Age 7 years. White male. High power, showing pus in Gasserian ganglion.

infection. The processes on both sides are certainly longer than a week, as given in the history.

SUMMARY

In reviewing the clinical histories in the light of the known pathology, they are chiefly remarkable for their paucity of symptoms which might have pointed toward the diagnosis of suppuration of the petrous pyramid. Three patients entered the hospital with meningitis. Of these, one had developed an abducens weakness ten days before entrance, before a simple mastoidectomy was done. This patient's x-rays of the petrosa revealed involvement of the pyramid. What significant symptoms the other two cases with meningitis might have presented before the onset of the meningeal infection is conjectural. One patient showed a profound picture of sepsis. In four patients nothing significant was noted. One patient had frontoparietal headache. One case had continued discharge since a simple

mastoidectomy five months before, and two revisions failed to reveal sufficient pathological change to explain the discharge. Another case also had continued discharge following the simple operation. X-ray findings of him showed involvement of the pyramid. None of the patients presented the classical picture of retro-orbital pain, continued or recurrent discharge, and low-grade sepsis.

Of the eleven cases eight were between four and nine years of age. The others were sixteen, twenty-four and thirty.

As to anatomy, two specimens, a bilateral case, show complete pneumatization of the apex. Three apices are diploetic and eleven mixed type. Of the mixed type, in six the marrow-containing cells predominate, in three the air cells are in the majority, and in two cases the air cells and the marrow cells of about equal amount. The completely pneumatized bones are from a patient of sixteen, while almost complete pneumatization is seen in a patient of six. Completely diploetic apices are seen in the youngest cases in the series, ages four and four and one-half years. Of the five bilateral cases, two are symmetrical and three non-symmetrical. Strangely enough all the unilateral cases are in left ears, but in three of the bilateral cases the right ear is more heavily involved than the left.

Time of illness, as taken from the histories varied from five days to nine months. Three cases, ill from five to seven days, entered moribund. In all of these the pathological study reveals processes considerably older than a week.

Routes of extension from the antrotympanic area to the apex could be traced in the serial sections in all but one case. Seven specimens show multiple routes of extension. In five specimens these were the superior route and hypotympanic cells; in one, superior route and peritubal cells. One apex was infected by the rupture of a tubal abscess directly into the marrow, as well as by hypotympanic cells in series with the apical marrow. All bilateral cases but one had identical pathways to the apex. Where there were multiple routes of progression they were not always of equal importance, and the more important route could be determined by the location of the more severe pathological changes. The unilateral route in five cases was by way of the peritubal cells, and in one case by the superior route; that is cells above, lateral and medial to the arcuate eminence, and cells proximal to the subarcuate fossa. In one case infection proceeded through the hypotympanic cells. As with most observers, our findings indicate that the superior route is the most frequent and important.

From the bacteriological standpoint, six cases were caused by the hemolytic streptococcus, one case by the streptococcus viridans, and one case by a streptococcus, type not stated. In two cases pneumococcus type I was found, and in one case a pneumococcus, type unknown. The type of pathological change, association of complications, etc., do not seem to be dependent upon the organism. Strangely enough, pneumococcus type III, claimed by some to be the chief offender in petrous suppurations, was not incriminated in any of these cases.

The pathological process in the apices is dependent upon the type of anatomy. Thus in the pneumatic apices it is an osteitis, in the diploetic apices an osteomyelitis, and in the mixed apices, except for two cases where a slight terminal infection in the pneumatic cells has not had time to infect the adjoining marrow, a combination of osteitis and osteomyelitis. Which one of these two predominates in any one case depends apparently, among other things, on the route of extension and the proportion of the two types of cells. The changes in the pneumatic cells are exactly comparable to those in the mastoid air cells. In some cases there may be chiefly swelling and infiltration of the mucosa with a minimum of free exudate. In other cases there is a severe suppurative reaction with pus cells predominant, and a further expression of this is destruction of cell walls and coalescent abscess formation. One has the impression that these apical cells are larger, have thinner walls, and are more prone to coalesce than mastoid cells. Likewise all variations and degrees of inflammation are seen in the marrow cells, including one or more types in a single case. In all specimens, except one, the infected pneumatic cell mucosa of the tympanum, peritubal cells or petrosal cells can be seen in contact with the marrow cells and this is certainly a mode of infection of the marrow. It is very likely that blood stream propagation also plays a röle. As a rule the marrow is more severely infected where it is in contact with the air cell, and the severity recedes as the distance from the infecting focus increases. The mildest type of infection is characterized by slight fibrosis of the marrow with a cellular infiltration consisting mainly of lymphocytes, plasma cells and histiocytes. Next is a more severe marrow fibrosis. This is the type of process seen frequently at a distance from the source of infection or at the edge of a suppurative focus, and is obviously a reparative, protective mechanism, an effort to limit the spread of disease. Though it is non-suppurative, still such a lesion will show bone resorption and new trabeculæ formation. A rather frequent process is one in which the marrow is replaced by a heavily

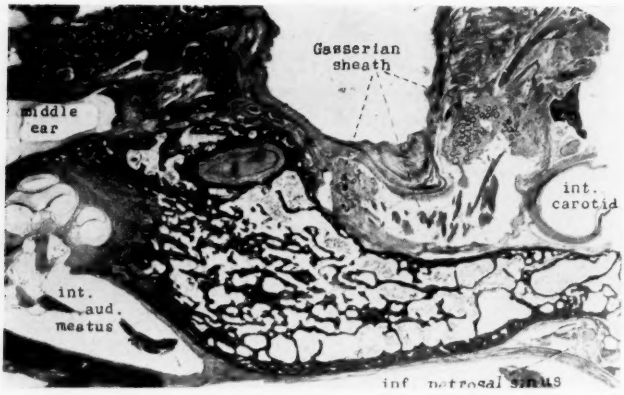

Fig. 17. No. 70 Left. Horizontal section. The Gasserian sheath as seen in the microscopic sections. Normal diploic apex.

infected, very vascular granulation tissue with a minimum of frank suppuration. Such a process apparently has great potentialities of bone destruction, and the apex is replaced by a coalescent area filled with granulations. Usually such bone destruction also involves the cortex with one or more pathological dehiscences. These granulomatous areas also show attempts at bone formation. Finally there is a frankly suppurative process where the area involved is a coalescent abscess, and this apical empyema, in its end-result is exactly similar to a coalescent abscess which follows the breakdown of pneumatic cells.

In the one case where no route of infection to the apex could be traced, the diploetic cells, of which it was composed, showed a fibrotic reaction with several small abscesses. This is essentially the lesion that Lindsay has recently emphasized. He feels that this represents a reactive, reparative, limiting process to an infective lesion in the temporal bone, which is not in continuity with the apex, and he does not believe it to be an osteomyelitis. With this we do not agree. It is not a suppurative lesion, but it is a response to an infection. Histologically it is identical with the marrow response which may occur in an infected apex, where the marrow is in contact with frank suppuration, and where the marrow and systemic resistance is such

as to react with a limiting fibrosis, instead of with suppuration. It is very different from the usual diploetic apical reaction to a neighboring infectious focus which manifests itself as a hyperplasia, and is probably what Eagleton has named the reactive osteomyelitis.

We have seen that the usual case is one of a mixed type of infection in a mixed type of apex. Both marrow and apical cells may contribute to a coalescent abscess or to a coalescent granuloma. Most observers have reported that the mixed type of apices are in greatest number. Moreover a wholly diploetic apical infection may result in that which is very similar to an infection in a pneumatic apex. In view of these pathological findings one questions very strongly the current belief that petrous suppurations clinically are to be divided into those occurring in a marrow containing apex, and those in a pneumatic apex, with different clinical syndromes, recognizable as such, and with different surgical approaches. Certainly in the usual run of cases it is not possible to say clinically how much is diploetic and how much pneumatic, whether it is wholly one or the other, and what symptoms, in a particular case are dependent upon the osteitis and what upon the osteomyelitis. In a few cases this subdivision is probably clinically correct. Thus in Case 31 of our series there is a phlegmonous osteomyelitis characterized clinically by a sepsis and pathologically by an acute infection with multiple venous thrombosis, and practically no body defense.

Further significant pathological findings were abscesses in the carotid sheath or subperiosteal abscesses in the carotid canal in six cases, in two of which the infection entered the canal through pathological dehiscences. In four of these cases there was present pericarotid thrombophlebitis, and two of these latter had resulted in cavernous sinus thromboses. Also present were three cases of suppurative labyrinthitis. Three instances of retropharyngeal abscess were present. In two it was by direct extension, and in one by retrograde venous thrombosis.

The fatal lesion was cerebellar abscess in two cases and meningitis in nine. The two cases of cerebellar abscess were the only ones where the greatest expression of disease of the petrosa was not in the apex.

The routes of extension to the meninges were by direct extension, vascular, and combinations of these two. In one instance direct extension was through the dura of the middle fossa and in two instances through the dura of the posterior fossa. One patient had

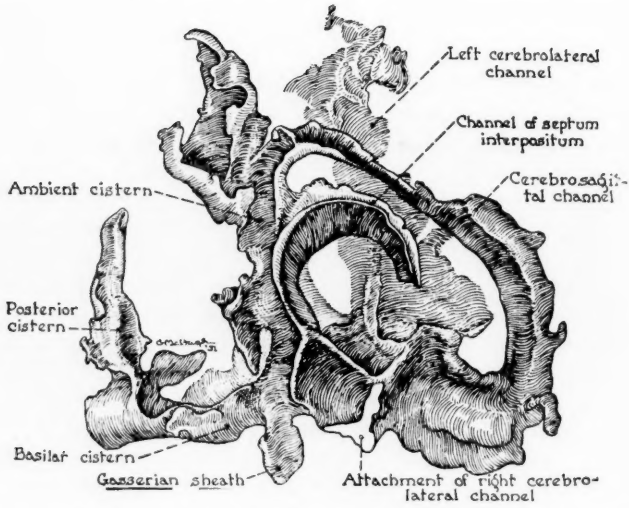

Fig. 18. Cast of the cisterns of cerebrospinal fluid (after Locke and Naffziger). Photographed from Bailey's intracranial tumors.

direct extension into the posterior fossa and into the middle fossa with pus in the Gasserian ganglion, and one had direct extension into the posterior fossa and infected thrombi in the Gasserian ganglion with an abscess of the ganglion. In Case 6358, bilateral, the infection on the right side extended into the posterior fossa directly, and the Gasserian ganglion on the left side was involved in infection both from a thrombosed vein, and by direct extension through the dura. In three cases the extension was most likely hematogenous in origin. This was indicated by pericarotid venous thrombosis, intradural infected thrombi, or pus in the Gasserian ganglion without direct extension through the dura. In one case no route could be traced, and it is likely that the meningeal soiling was not from the apex.

The work of Pietrantoni, as quoted by Ruskin, offers a logical understanding of the hematogenous pathways to the Gasserian ganglion and intracranial contents. This observer, studying injected temporal bones, noted that "between the anterior third of the tym-

panic cavity and the tip of the petrous bone there is a tract of compact bone tissue traversed by the carotid canal. This tract of bone is traversed by a fine, reticulated plexus of veins, which externally is continuous with the submucous venous plexus of the tympanic cavity, and internally joins the many venous trunks of the tip of the petrous bone. The posterior ones of these empty into the inferior petrosal sinus a little before it unites with the cavernous sinus, the median ones anastomose with the pericarotid venous plexus, and the anterior ones, having united with the vascular plexus which surrounds the Gasserian ganglion, empty into the cavernous sinus." Numerous slides in this collection reveal these small veins penetrating the cortex underneath the Gasserian ganglion. In eight cases abscesses of the Gasserian ganglion were seen. In two of these the extension was directly through the dura from the infected apical contents, although in six other instances, the very resistant nature of the dura was seen, for though it was in contact with infection below, it was not involved except at the point of contact. In six cases, however, there was pus in the Gasserian ganglion without direct extension demonstrable through the dura. In all these cases there was evidence of a thrombophlebitic process either of the veins of the Gasserian ganglion or else of the pericarotid venous plexus. It is through these veins, by way of the routes stated by Pietrantoni, that we believe infection reaches the Gasserian ganglion with intact dura.. This route is of great importance. It is further attested to by the fact that in the cases of meningitis of this series, as well as the instances of purulent meningitis studied microscopically in this laboratory, abscesses of the Gasserian ganglion were seen only where direct extension was demonstrable, or where there was microscopic evidence of a thrombophlebitis in the apical region. Pus was not observed in the Gasserian ganglion in the presence of a meningitis per se.

Further we regard abscesses of the Gasserian ganglion as of special significance in the production of meningitis. Although this ganglion lies in the cavum Meckelii, between two layers of dura mater, when pus has reached it, the situation is different from that present with the usual extra-dural abscess, even though intact dura over the ganglion is between pus and meninges. The explanation is found in the accompanying diagram of the subarachnoid space and basal cisterns (Fig. 18). A subarachnoid cistern projects laterally between leaves of dura and envelops the Fifth nerve and the Gasserian ganglion (Fig. 17). Therefore pus in the Gasserian ganglion, although anatomically extradural, in reality is in contact with the subarachnoid space, and infection of the latter easily follows.

Through this anatomical route pus could possibly enter the Gasserian sheath retrograde from a diffuse meningitis, but as already stated, this has not been observed.

55 East Washington St.

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LXXXI

OPTIC NERVE COMPLICATIONS OF ACCESSORY NASAL SINUS DISEASE*

ISRAEL STRAUSS, M.D.
AND
WILLIAM NEEDLES, M.D.
NEW YORK

INTRODUCTION

The neurologist may be forgiven if it is with some trepidation that he ventures to discuss what is essentially an ophthalmologic complication of a rhinologic disorder, since certain aspects of the problem fall within the scope of, and can best be evaluated by, the specialist in these respective fields. However, the neurologist, because he is not an ophthalmologist nor a rhinologist, may be able to view the subject with somewhat more detachment, free from the bias which has, as a rule, placed these specialists in two opposing camps. The fact that the condition under discussion must frequently be differentiated from various neurological diseases, notably multiple sclerosis, provides another reason why the neurologist must concern himself with the problem.

The cases of rhinogenic optic neuritis that we are about to discuss present a syndrome of suddenly appearing blindness, frequently preceded by an upper respiratory infection. The most painstaking examination fails to disclose any adequate cause for the condition except that the sinuses are consistently found diseased. Surgical intervention on the sinuses is often followed by prompt, and in some cases, dramatic improvement in vision. This sequence of events has seemed to many a fairly cogent argument for a casual relationship between the disease of the sinuses and the affection of the optic nerve. Others, however, dispute vigorously such a conclusion.

A systematic account of the development of the subject will not be attempted here since it can be found in other papers dealing with this theme.[1]

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*From the Neurological Service of Dr. Israel Strauss at the Mount Sinai Hospital, New York City.

Presented before the New York Neurological Society, April 5th, 1938.

The controversial state of the problem is reflected in the widely divergent statistics regarding its frequency. Thus Grosz,[2] who observed about 100 cases of retrobulbar neuritis annually, thought that approximately 15 per cent could, with reasonable certainty, be ascribed to diseased sinuses. Wilmer found an incidence of 11 per cent in his cases, Walker 4 per cent, Langerbeck 3.5 per cent, and Sheerer and Maier 1.5 per cent.[3] Traquair[4] on the other hand stated "in Edinburgh we have come to the conclusion that rhinogenic retrobulbar neuritis is a very rare condition." Behr[5] expressed skepticism regarding the existence of any such entity as retrobulbar neuritis of sinus origin. E. V. Hippel[6] appears equally skeptical, while Lillie[7] affirms "sinusitis is the last etiologic factor I should consider in a patient with acute retrobulbar neuritis." In his series of 225 cases, only one was considered attributable to diseased sinuses. Those who cite higher incidences doubtless are regarded by the others as too lenient in the criteria they set up.

While the disease may be slow and insidious in onset it usually sets in with a sudden, alarming diminution in vision. This is generally unilateral, less often bilateral. Not infrequently there is an accompanying upper respiratory infection. Periorbital pain, tenderness on pressure over the eyeballs and pain upon movement of the eyeballs are common symptoms. Visual acuity is impaired to varying degrees. In typical cases a central scotoma is found, accounting for the frequent subjective complaints of a black spot in front of the eye; the central scotoma is merely for color in the milder cases, for form as well as color in the severer cases. The scotoma may spread peripherally so that only a small zone of annular vision is retained. In some cases an annular peripheral scotoma is found. Van der Hoeve[8] considers characteristic of retrobulbar neuritis not merely the central scotoma, but also enlargement of the blind spot (Van der Hoeve's sign). The latter he attributes to involvement of the peripapillary fibers, shown by Fuchs to supply the portion of the retina adjacent to the optic disc and to lie at the periphery of the optic nerve. It should be stated, however, that others regard a slight enlargement of the blind spot as often merely a symptom of fatigue. If the pathologic process confines itself to the macular fibers in the retrobulbar portion of the nerve, the fundus, in the acute stage of the disease, at any rate, appears entirely normal. If, however, the process extends forward and involves the nerve head, one observes the picture of optic neuritis with reddening of the disc, blurring of its margins, narrowing of the arteries, engorgement of the veins, and in some cases, hemorrhages

and exudates. Cushing to the contrary, others report a fundus picture in such cases which is indistinguishable from that of choked disc. The pupil of the affected eye is usually larger than the other and responds less fully to direct illumination while its consensual response is intact.

A great many cases recover spontaneously. In these as well as in those in which the process subsides after surgical intervention there may be complete restitution of function with no evidence in the fundus of any antecedent disease. In other less fortunate cases optic atrophy supervenes and is discernible by pallor of the disc or irregularity of its margins.

The sinuses generally held responsible are the sphenoidal and the ethmoidal, particularly the posterior cells of the latter group. These sinuses are in intimate contact with the optic canal. It will be recalled that the optic nerve, after coursing for a distance of about one centimeter intracranially, runs for approximately five millimeters through the optic canal to continue in the orbital cavity for another three centimeters. Because of the intimate relationship between the sinuses and the nerve it is apparent that infection may readily spread from one to the other. If disease of the frontal or maxillary sinuses produces a rertobulbar neuritis, the mechanism is considered a distance effect, akin to that obtaining when infected tonsils, for example, are the responsible agent. Considerable difference of opinion exists relative to the significance to be attached to various forms of pathology found in the sinuses, but among those observers who maintain the rhinogenic origin of optic neuritis there is practically unanimity of opinion regarding one point, namely, that the evidence of disease in the sinuses need not be imposing or formidable. The sinus condition is generally of so little concern to the patient that it is almost invariably the ophthalmologist who is first consulted. It is rare in cases of retrobulbar neuritis of supposedly rhinogenic origin to find a frankly purulent sinus. It has, on the other hand, been found that what may be regarded as relatively trivial involvement of a sinus may lead to serious alterations in the optic nerve.

We present now a series of 14 cases, observed at the Mount Sinai Hospital, the study of which has helped determine our own standpoint in this thorny problem. The first group consists of fairly typical instances of rhinogenic optic neuritis; the second, of cases in which multiple sclerosis had to be seriously considered;

and, finally, one case which we think can best be described as sphenoiditic hydrocephalus.

REPORT OF CASES

CASE 1.—N. R., a tailor, aged 45, entered the Mount Sinai Hospital, September 30, 1936, and was discharged October 31, 1936.

Past History: Appendectomy 14 years ago. Pulmonary tuberculosis seven years ago.

Present Illness: Two weeks before admission the patient had an upper respiratory infection. One week later he developed pains in both eyes, and dimness of vision, more marked on the right. His vision became progressively worse.

Physical Examination: The patient appeared chronically ill. His eyeballs were tender to palpation. Movement of the eyeballs also induced pain. There was tenderness over the left ethmoid. A postnasal discharge was present. Dullness on percussion and rales were found over the upper portion of the posterior aspect of the right chest.

Neurological examination was negative except for the ocular findings. Visual acuity was 3/200 in the left and 1/200 in the right eye. A central scotoma was present bilaterally. The fundi showed reddish discs with blurred margins, more marked on the right. In the left fundus three small hemorrhages could be seen above the disc. The diagnosis was optic neuritis.

The cerebrospinal fluid was negative.

Since clinical and radiographic investigation of the sinuses disclosed widespread disease, a bilateral spheno-ethmo-antrotomy was performed on September 30.

Three days later visual acuity was 5/100 in the right, 10/100 in the left eye.

The sinuses were irrigated on several occasions, and typhoid vaccine administered. There was progressive improvement, so that before discharge visual acuity was 20/50 plus 2 in the right, 20/32 in the left eye. The fields showed accompanying changes (see Figs. 1, 2, 3). The optic discs showed some degree of temporal pallor.

When seen at the follow-up clinic two months after discharge, the patient was complaining of headaches. He was still being treated for sinusitis. His vision was said to be "poor," but since no quantitative estimation was recorded, it is impossible to state how much significance should be attached to this observation. He failed to report for further study during the next year.

Comment: This case illustrates several features of optic neuritis from diseased sinuses which are quite typical of the syndrome: the onset following an upper respiratory infection; the pain and tenderness of the eyeballs; the pain induced by movement of the eyeballs; the marked impairment of visual acuity with the presence of a central scotoma; frank evidence of pathology in the sinuses; eradication of the foci of infection, with prompt improvement in the visual status.

Whether this patient subsequently had another flare-up in his condition cannot be decided since he failed to report for further observation.

CASE 2.—E. S., a housewife, aged 44, entered the Mount Sinai Hospital, May 12, 1937, and was discharged May 27, 1937.

Past History: Irrelevant.

Present Illness: Four weeks before admission after a mild upper respiratory infection, the patient noted a black spot in the visual field of the right eye.

Physical examination: The right ear drum was thickened. The tonsils were enlarged and there was a slight postnasal drip. There was slight tenderness over the ethmoid sinuses bilaterally.

Neurological examination was negative except for the ocular findings. Visual acuity was 20/32 in the left eye and 20/38 plus 4 in the right. The left fundus was normal. The right disc was quite red, with most of the capillaries engorged, and the pit barely visible. All the margins were obscured by edema, which was least marked temporally. A fine gray exudate was interspersed in the edematous peripapillary area. Above the disc was a fine hemorrhage; below were tiny hemorrhages. The arteries near the disc were thin, the veins full and dark. The elevation measured barely one diopter. Visual field studies showed an enlarged blind spot and a constricted field for color (see Fig. 4). The diagnosis was optic neuritis.

Lumbar puncture showed a clear fluid, under a pressure of 80 mm. H_2O. The Pandy reaction was 2 plus. There were 2 cells. The total protein was 42 mg./100 cc. The Wassermann reaction and the colloidal gold curve were negative. The urine and the blood count were normal.

Attention was directed toward the sinuses. This disclosed a low-grade hypertrophic ethmoiditis and antritis. Flakes were obtained on irrigation of the right sphenoid. The tonsils were also found diseased. X-ray showed clouding of the antral, ethmoidal and sphenoidal sinuses.

A right spheno-ethmoidectomy was performed May 17 with the disclosure of a polypoid mucosa in the ethmoidal cell, with some thick mucoid secretion. The sphenoid sinus proved negative. Subsequently the sinuses were irrigated.

Two days postoperatively vision was subjectively and objectively improved and the changes in the fundus were noted as subsiding. Five days following the operation vision in both eyes was 20/32. The field for central color vision was enlarged (see Fig. 5). Two days later vision in the right eye was 20/32 and in the left 20/25 plus 3.

A follow-up note June 24, 1937, reads, "No complaints." The fundi were normal.

Comment: In this case an upper respiratory infection again preceded the onset of visual disturbances. Clinically and radiographically there was evidence of disease of the accessory nasal sinuses.

The scotoma which was subjectively present at the onset could no longer be detected at the time of observation, and visual acuity was not profoundly impaired. The changes in the fundus were quite marked, however. Operation was followed by improvement in the visual acuity, visual fields and in the fundus picture.

There was no evidence of disseminated lesions of the nervous system, and the age of the patient, as well as the exudative and hemorrhagic changes in the fundus, militated against the diagnosis of multiple sclerosis.

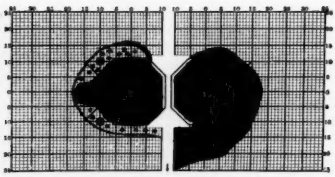

Fig. 1. Case 1. Four days after operation. ½ mm. object.

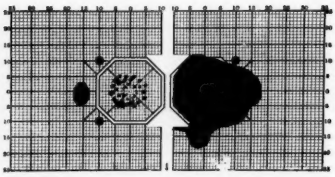

Fig. 2. Case 1. 18 days after operation. 1 mm. object.

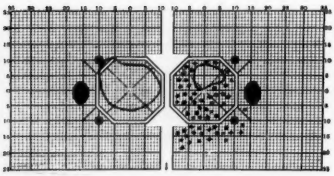

Fig. 3. Case 1. 26 days after operation. 1 mm. white, 2 mm. red object.

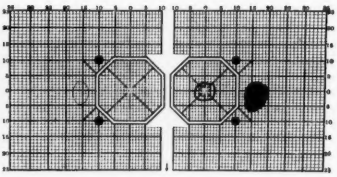

Fig. 4. Case 2. One day before operation.

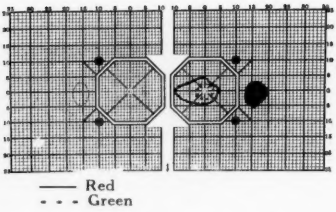

Fig. 5. Case 2. Seven days after operation.

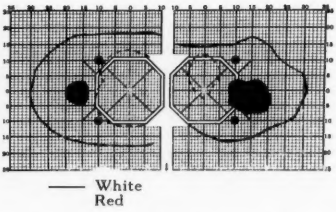

Fig. 6. Case 3. 24 days after operation.

CASE 3.—L. N., a schoolboy, aged 9, entered the Mount Sinai Hospital, August 3, 1937, and was discharged September 12, 1937.

Past History: Except for an attack of sinusitis at the age of 3, and frequent colds since then, the past history was negative.

Present Illness: One week before admission the patient awoke, complaining of pain in the right eye. The next day his eyelids appeared inflamed and he had a running nose. The following day vision was greatly impaired in the right eye and the day thereafter there was allegedly no vision in that eye. Two days before admission he developed pain and impaired vision in the left eye.

Physical examination showed a postnasal drip, tenderness over the sinuses, tenderness on palpation of the eyeballs, and pain on movement of the eyes.

The neurological examination was negative except for the ocular findings. There was light perception in the right eye at a distance of one foot, and recognition of fingers at two inches. Visual acuity in the left eye was 20/175 plus 1. Perception of colors was absent in the right eye, and markedly impaired in the left. Both discs were hyperemic with blurred margins, especially nasally and superiorly. There was an elevation of about one-half diopter in each eye. The veins were engorged. A diagnosis of bilateral optic neuritis was made.

The spinal fluid was clear. The initial pressure was 135 mm. H_2O. The Pandy test yielded a faintly positive result. There were 45 cells, of which 94% were mononuclear in type. A culture of the fluid yielded no growth.

Attention was directed to the state of the accessory nasal sinuses. An x-ray of the sinuses showed slight cloudiness of the left ethmoid cells and slight haziness in outline of the left sphenoid.

A bilateral spheno-ethmoidectomy was performed August 4. The right sphenoid was reported as showing slight thickening of the mucous membrane. Subsequently the sinuses were irrigated. For a day or so the patient showed meningeal signs. Another spinal fluid examination showed the presence of 17 cells, 16 being of the mononuclear type.

The patient was given inoculations of typhoid vaccine.

He showed a progressive improvement of the fundi, visual acuity, and visual fields (see Figs. 6 and 7). An examination of the fundi one month after admission showed merely slight edema at the margin of each disc. The visual acuity at the time of discharge was 20/20 minus 2 in the right eye; 20/15 minus 2 in the left eye.

A follow-up note of May 5, 1938, reads: "Eyes are being looked after by own physician. Feels well except for prolonged running nose when he gets a cold."

Comment: This case in which the vision of a very young individual seemed gravely imperiled, yielded ultimately a very gratifying result.

The occurrence of a cold at the onset is again noteworthy, suggesting, as it does, an infectious process as the basis of the entire condition.

The meningeal syndrome that supervened was for a time disconcerting, but it, too, rapidly cleared up.

The youth of the patient is worthy of comment, since it is generally recognized that optic neuritis is an uncommon event in childhood, and the disparity

between the frequency of sinusitis in childhood and the rarity of optic neuritis has been employed by some as an argument against any causal relationship between the two.

In the present case, at any rate, measures directed against the suspected sinuses were followed by a fairly prompt and very complete subsidence of symptoms.

There was no evidence of disseminated lesions of the nervous system, nor did the patient fall within the usual age group for multiple sclerosis

CASE 4.—E. R., a schoolgirl, aged 15, entered the Mount Sinai Hospital, October 26, 1936, and was discharged December 17, 1936.

Past History: One year ago the patient suffered an attack of sinusitis.

Present Illness: Six weeks before admission, while viewing a cinema, the patient happened to cover her left eye and noted that vision was poor in the other eye. Since that time her vision had improved. The patient's eyesight had been tested two years previously in the course of a routine examination and been found satisfactory.

In the clinic of the hospital, 10 days before admission, a central scotoma in the right eye had been detected.

Physical examination showed a postnasal discharge.

Neurological examination was negative except for the ocular findings. Visual acuity was 10/68 minus 3 in the right eye, 20/38 minus 3 in the left. The right optic disc appeared reddish, with slight blurring of the nasal margin, and distention of the veins. The left fundus showed a reddish disc, but was otherwise normal. These findings were taken to indicate a retrobulbar neuritis, with extension forward to involve the discs.

The cerebrosphial fluid was negative in all respects.

Examination of the sinuses disclosed bilateral ethmoiditis and antritis. X-ray of the sinuses was negative.

A right spheno-ethmo-antrotomy was performed November 10 and thickening of the membranes of the sphenoid and postethmoidal cells was found. There was no pus.

Subsequently, the sinuses were irrigated and typhoid vaccine administered.

November 14, vision in the right eye was 15/200; a central scotoma for color was present (see Fig. 8).

November 21, vision in the right eye was 10/122.

November 26, vision in the right eye was 10/137 plus 1, and in the left 20/68.

November 30, vision in the right eye was 10/122 plus 1, and in the left 20/77.

Since the left eye was becoming worse, a left spheno-ethmo-antrotomy was performed December 4. Improvement in both eyes occurred, so that shortly before discharge vision in the right eye was 20/86, that in the left 20/32 minus 2 (see Fig. 9).

A follow-up note, February, 1937, stated that after a period of improvement in vision, the patient again was complaining of some visual impairment.

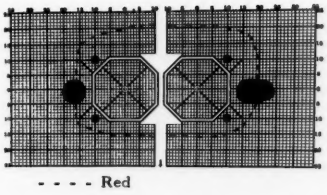

Fig. 7. Case 3. 38 days after operation.

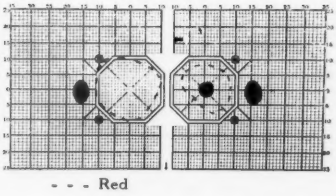

Fig. 8. Case 4. Six days after first operation.

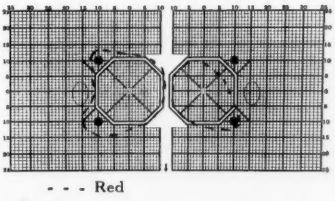

Fig. 9. Case 4. Two months after first operation, one week after second operation.

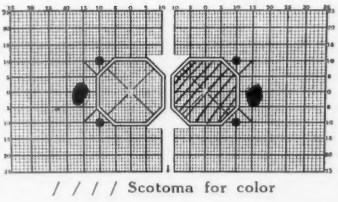

Fig. 10. Case 7. Showing the central scotoma operation for color.

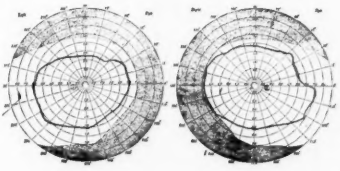

Fig. 11. Case 7.

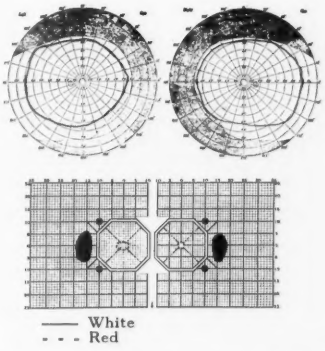

Fig. 12. Case 8. Thirteen days before operation.

The fundi were unchanged. Efforts to have the patient report on September 1, 1937, and January, 1938, were unsuccessful.

Comment: In this case there was manifest evidence of disease of the sinuses, associated with the signs of an optic neuritis and a sinusectomy was on two occasions followed by improvement in vision. We think that tentatively, at least, it is justifiable to regard the case as one of the optic neuritis secondary to infection in the sinuses. Further observation is, of course, desirable, since, as is well known, multiple sclerosis may be ushered in by a retrobulbar neuritis and may remain monosymptomatic for many years thereafter.

CASE 5.—R. A., a housewife, aged 49, entered the Mount Sinai Hospital on April 19, 1937, and was discharged May 6, 1937.

Past History: The patient had a choleocystectomy and appendectomy five years ago.

Present Illness: Two weeks before admission the patient suddenly developed impairment of vision in the left eye and headache in the frontal region. The visual impairment progressed and the headache persisted up to the time of admission.

Physical examination was negative except for the presence of a laparotomy scar and diseased tonsils

The neurological examination was negative except for the ocular findings. The visual acuity of the right eye was 20/70 minus; with the left eye shadows were perceived at a distance of three feet. The optic nerve on the right was yellowish in color; its margin was indistinct, especially nasally; the temporal edema was confined to a striation and haze in the retina extending into the macula. Beyond the macula was an area of swollen retina. The veins were full and the arteries narrowed. In the left eye the picture was similar, but more marked. The nerve head was more blurred, the macula had almost opaque striations due to swollen fibers and exudation.

The blood Wassermann was negative. The urea and glucose of the blood were normal.

Because of past experience attention was promptly directed to the sinuses. An x-ray of the sinuses showed slight clouding of the ethmoid cells bilaterally. The right antrum was smaller than the left, possibly due to thickening of the membrane.

The patient was considered to have an optic neuritis and retinitis due to pathology in the sinuses.

Her vision continued to get worse, measuring 20/100 in the right eye, with total loss of vision in the left. Surgical intervention was recommended, but the patient insisted on leaving the hospital against advice.

Comment: This patient presented a more advanced form of optic nerve involvement from diseased sinuses, in which the process did not confine itself to the retrobulbar portion of the nerve, but spread forward to involve the nerve-head and the adjacent retina. In this case there was involvement of both optic nerves instead of the more common unilateral involvement. In the absence of any other factors to account for the condition and with the evidence of diseased sinuses unequivocal, the diagnosis of a rhinogenic optic neuritis seemed the most plausible. The advanced age and pronounced fundus changes rendered the

diagnosis of multiple sclerosis very unlikely. Unfortunately this patient would not submit to exploration of her sinuses, which appeared to us imperative in view of the progressive failure of vision, and her subsequent fate remains unknown.

CASE 6.—R. B., a painter, aged 46, entered the Mount Sinai Hospital, March 12, 1925, and was discharged April 2, 1925.

Past History: The patient had been a painter for 30 or more years, but had never had any symptoms of plumbism.

Present Illness: One and a half years before admission he suddenly noted visual impairment in the right eye. Objects were seen only at close range, colors were not recognized. One-half year later he had a nasal operation performed, type unknown, without obtaining any relief. During the past two months there had been visual impairment of the left eye also. In the clinic, shortly before admission to the hospital, vision was found to be 20/40 in the left eye, with recognition of fingers at five feet in the right. The fundus picture suggested postneuritic optic atrophy. An x-ray of the sinuses showed cloudiness of both frontal and ethmoid sinuses, with a suggestion of antral involvement.

Physical examination negative except for cryptic tonsils.

Neurological examination was negative except for the ocular findings. Vision was 10/200 in the right eye, 20/50 in the left. The margins of both discs were blurred, the discs were pale, and the arteries thin. Again the impression was that of a postneuritic optic atrophy.

The Wassermann of the blood was negative. The cerebrospinal fluid was negative in all respects.

The case was considered one of optic neuritis secondary to involvement of the sinuses. The tonsils were removed and the patient referred back to the eye and nose and throat clinics for further observation.

Comment: Perhaps this case would have been treated less conservatively if it had been observed more recently than 13 years ago. The presence of optic atrophy most likely prompted observers to withhold further operative measures. In the experience of some, however, relief has been obtained by sinusectomy even after signs of optic atrophy have made their appearance.

Perhaps the outcome in this case would have been more satisfactory had less time elapsed between the onset of the illness and the initial attempts at therapy.

Multiple sclerosis looms up so prominently as a diagnostic possibility in every case of retrobulbar neuritis that it deserves special consideration. As is well known, retrobulbar neuritis may be the first signal of the disease and may precede the appearance of other symptoms by a decade or more. Its onset in multiple sclerosis may be accompanied by pain about the eyes, as is the case in retrobulbar neuritis of rhinogenic origin. Similarly, in multiple sclerosis the pathology may not be limited to the retrobulbar portion of the nerve, but in some instances extends forward to involve the papilla, producing a picture resembling optic neuritis. In rare cases the findings in the fundus have been indistinguishable from

papilledema. Of course, when the symptoms and signs of retrobulbar neuritis supervene in an already well-established case of multiple sclerosis, or when the current neurological examination discloses, in addition to retrobulbar neuritis, evidence of disseminated involvement; or when, finally, continued observation of a case witnesses the gradual unfolding of signs typical of the disease, the diagnosis appears obvious. Yet even when the picture of multiple sclerosis is well established difficulties arise when one considers that it is possible for a patient with multiple sclerosis to develop sinusitis and, secondarily, an affection of the optic nerve. Or conversely, that in cases in which an evanescent retrobulbar neuritis antedates by years the onset of a syndrome of multiple sclerosis, the original symptom might have been rhinogenic in origin and totally unrelated to the multiple sclerosis that subsequently appears. It is well in this connection to recall the dictum, attributed to Marburg, that while multiple sclerosis must be considered as a possibility in every case of retrobulbar neuritis, not every case of retrobulbar neuritis of obscure etiology is one of multiple sclerosis.

We present now some cases bearing on this phase of the problem.

REPORT OF CASES

CASE 7.— H. G., aged 17, was first seen by Dr. Israel Strauss on November 14, 1935.

Past History: Double mastoidectomy at the age of 18 months. Tonsillectomy at two and one-half years. Whooping cough, measles, mumps in childhood.

Present Illness: Several weeks prior to observation the patient developed a mild headache over the right eye, which lasted about three days. Ten days before observation blurring of vision in the right eye occurred. Examination of the eyes at that time showed a central scotoma in the right eye, with remains of peripheral vision and recognition of objects at a distance of one and one-half feet. There was pallor of the temporal half of the right optic disc. Examination of the sinuses was reported as showing a left ethmoiditis. The patient was referred to Dr. Strauss by Dr. J. W. Smith for the purpose of ruling out multiple sclerosis.

Neurological Examination: The deep reflexes were extremely hyperactive. The abdominal reflexes were absent. There was an equivocal Babinski sign on the right and a tendency to dorsoflexion of the big toe on the left. The optic discs appeared pale. Dr. Strauss suspected that the condition might be one of multiple sclerosis, but suggested nevertheless that the sinuses be explored.

Dr. Rudolph Kramer performed a right spheno-ethmoidectomy November 15, 1935. Clumps of pus were found in the right sphenoid. In addition there was a recess with an inflamed mucous membrane extending beneath the floor of the sella turcica.

Vision improved after the operation. Gradually, however, the blurring recurred. Neurological examination March 3, 1936, showed definite atrophy

of the right optic nerve and a central scotoma. Only the right upper abdominal reflex was faintly elicited. The right Babinski sign was again equivocal.

The patient was admitted to the Mount Sinai Hospital, March 9, 1936, for a course of fever therapy and was discharged March 26, 1936. While there a visual field study showed a central scotoma for color in the right eye (see Fig. 10).

April 3, 1936, the patient was reported much improved. A course of eight injections of bismogenol was resorted to.

In June the visual acuity of the right eye, which in March, 1936, had measured 15/200, was now 15/15 minus 4. Vision was not entirely clear, however. The blind spot was enlarged. There was no scotoma. The temporal pallor of the discs persisted. The knee and ankle jerks were hyperactive, but there was no Babinski sign. Quinin and vitamin therapy were prescribed.

September, 1936, all the abdominal reflexes except the left lower were present. There was no Babinski sign. The optic nerves appeared improved. Vision in the right eye was 20/20. There was no enlargement of the blind spot and no central scotoma. September, 1937, the ocular findings were reported as unchanged. Perimetric examination showed a 15-degree contraction temporally and 10-degree supero-nasally for form, a definite improvement over the status in November, 1936; in December, 1937, the status was again unchanged. Perimetric examination at that time (see Fig. 11) showed the same constriction in the peripheral fields which had been present during the preceding year.

Comment: This patient fell within the age group of multiple sclerosis. She presented, moreover, definite evidence of disseminated involvement of the nervous system with characteristic fluctuation of signs. The neurologist concluded that the patient had multiple sclerosis. Yet, because of the diseased state of the sinuses, he felt justified in urging operation. Following the operation improvement occurred. Later there was a recurrence of visual difficulties, with, again, improvement following fever therapy. The question arises whether the improvement following sinusectomy was merely coincidental or, whether, on the other hand, the eradication of an infectious focus did not play a substantial part in producing the favorable result. Did the infectious process in the sinuses render the optic nerve more vulnerable to the multiple sclerotic process or, conversely, did the multiple sclerosis render the optic nerve more susceptible to the effects of the infectious agent in the sinuses? These are questions we raise, but cannot answer.

CASE 8.—P. R., a housewife, aged 31, entered the Mount Sinai Hospital, March 31, 1937, and was discharged April 29, 1937.

Past history was irrelevant.

Present Illness: Four weeks ago, coincidental with the onset of her period, the patient developed pain in the right eye, followed two weeks later by profound impairment of vision in the left eye. An ophthalmologist who saw her at the time reported vision as 20/40 in the right eye, 20/200 in the left. Two days later vision was 20/40 in both eyes.

Physical examination was negative.

Neurological Examination: Visual acuity was 20/45 in the right, 20/40 in the left. Color perception was impaired. There were bilaterally enlarged blind spots (see Fig. 12). The right disc was slightly pink and the margins

hazy. The elevation was one and one-half to two diopters. The veins were full and the arteries thin. The left fundus presented a similar picture with an elevation of one diopter. The right pupil was greater than the left, both reacted sluggishly to light. There was tenderness of the eyeballs to pressure. There was a right central facial weakness. The right biceps and patellar reflexes were greater than the left. Bilateral Hoffmann signs were present and the left abdominal reflexes were diminished.

The sinuses were investigated. Both ethmoids and the right sphenoid and antrum were found infected.

The cerebrosphial fluid was negative.

The diagnosis was considered to lie between multiple sclerosis and optic neuritis of sinus origin.

Vision became worse: 20/85 in the right eye and 20/45 in the left. Central color vision was impaired (see Fig. 13). A few days later vision was 20/97 minus 2 in the right eye, 26/60 minus 3 in the left, and a day after that 20/122 in the right, 20/80 plus 1 in the left.

A right sphenoidectomy was performed April 14, followed two days later by a left sphenoidectomy. Promptly the changes in the fundi regressed. Vision became 20/32 plus 3 in the right eye, 20/25 minus 6 in the left eye. The fields are shown in the accompanying Fig. 14.

The patient was discharged as a multiple sclerosis suspect despite the improvement in vision following the sinusectomy.

When observed at the follow-up clinic two months later, the patient gave a history of stiffness and numbness in the left hand during the two preceding weeks. There was weakness of the left arm, ataxia in the upper limbs, absence of abdominal reflexes, the left knee jerk was greater than the right, and the Babinski sign was positive on the left. The optic discs showed secondary optic atrophy.

Comment: The diagnosis of multiple sclerosis appears fairly well established in this case. If this is granted, was the improvement in vision after operation merely a coincidence, or was there, in addition, an optic neuritis secondary to disease of the sinuses?

CASE 9.—G. B., a machine operator, aged 19, entered the Mount Sinai Hospital, February 7, 1932, and was discharged February 17, 1932.

Past history was irrelevant.

Present Illness: About 10 days before admission the patient experienced mild pain in the left eye on gazing toward the left and there was blurring of vision at the center of the left field of vision. Four days before admission she noted slight nasal obstruction.

Physical examination was negative.

Neurological examination was negative except for the ocular findings. The fundi appeared normal. Visual field studies showed a small scotoma for form and color in the central portion of the field on the left.

Examination of the sinuses, directly and radiographically, disclosed no significant abnormalities. The tonsils were described as hypertrophic, cryptic and filled with pus.

The spinal fluid was normal in all respects.

A diagnosis of retrobulbar neuritis was made.

Five days after admission visual acuity in the right eye was 20/30 and in the left 20/40. The patient stated that her vision was improving. Red and green were identified, but blue was called "green." Four days later there was no evidence of any scotoma.

A follow-up note on May 26, 1932, stated that the patient was free of complaints. Vision was intact and the fundi was negative.

In June, 1934, the patient, since married, gave birth to twins after a normal, uneventful pregnancy.

She was admitted to the hospital for the second time April 24, 1935, and remained until May 18, 1935.

Present Illness: Three weeks ago she developed pain over the right eye. The following day she noted blurred vision in that eye, which increased up until a week ago, and then improved somewhat. In the clinic on April 13, vision in the right eye had been 20/50, in the left 20/20. A week later there was only peripheral vision left in the right eye.

Physical examination was negative.

Neurologic Examination: The left pupil was larger than the right. The right reacted very poorly to direct stimulation by light. There was slight narrowing of the right palpebral fissure. The Babinski sign on the left was considered positive and the abdominal reflexes occasionally were not obtainable on either side. The patient distinguished fingers at a distance of five feet at the periphery of the field of the right eye. The visual acuity of the left eye was 20/20. The left fundus was normal. The right showed blurring of the margin of the disc was one diopter elevation. The veins were full and the arteries narrowed. Visual field examination showed a large central scotoma, absolute in the right eye (see Fig. 15). The ophthalmological diagnosis was optic neuritis. In the opinion of the neurologist, multiple sclerosis was to be considered, but a neoplasm of the right frontal lobe had to be excluded.

Lumbar puncture disclosed spinal fluid at an initial pressure of 90 mm. H_2O, no cells, a total protein of 55 mg./100 cc. and negative serology.

Rhinologic examination disclosed diseased tonsils, a Thornwald abscess and bilateral ethmoid and antral disease. An x-ray showed very slight clouding of the left antrum. If no other cause for the optic neuritis could be detected, a tonsillectomy and an operation on the sinuses were recommended.

On May 6, definite improvement in vision was noted and the visual field changes were much less marked.

On May 7, a tonsillectomy was performed and the Thornwald abscess opened.

Vision continued to improve so that on May 17 the visual acuity of the right eye was 20/24 and there remained only a tiny scotoma for color in the central field (see Fig. 16). The fundus also showed signs of regression. The only changes noted were thinning of the arteries in the right fundus and some blurring of the nasal margin of the disc with one-half diopter elevation.

The patient failed to report for follow-up on two occasions, so that no further data are available.

Comment: This case illustrates very well the need for prolonged observation of these patients before any definite conclusions can be drawn. On the first admission the patient presented a fairly classical picture of retrobulbar neuritis, whose etiology remained obscure. Because of the youth of the patient, multiple sclerosis had seriously to be considered, despite the absence of any signs of dissemination, and particularly in view of the negative findings in the investigation of the sinuses.

The patient remained well for more than three years, and significant from the point of view of a possible multiple sclerosis, went through a normal pregnancy during this period.

On her second admission the patient showed, in addition to affection of the optic nerve, a few other neurologic signs. These were few, but sufficiently definite to arouse the suspicion of a frontal lobe neoplasm, which diagnosis, in the light of the subsequent course, was discarded. The visual impairment began to clear up prior to any measures directed against foci of infection. And while this in itself does not rule out an infectious etiology, it also is consistent with the diagnosis of multiple sclerosis, which now appears most likely.

CASE 10.—S. E., a saleslady, aged 20, entered the Mount Sinai Hospital, August 31, 1930, and was discharged November 9, 1930.

Past history was irrelevant.

Present Illness: A week before admission the patient developed pain in the right eye and swelling of the lids. The following day there was pain in both eyes and vision in the left eye appeared dim. Soon thereafter vision in the right eye disappeared completely. Two days before admission she was observed at another hospital, where bilateral choking of the discs was reported, with absence of vision in the right eye and visual acuity of 20/30 in the left. An x-ray of the sinuses at the time showed clouding of the sphenoid sinuses, and a sphenoidectomy was performed. The sphenoid sinuses were found denuded of membrane; the posterior ethmoid cells were soft and necrotic. Following the operation vision in the left eye was almost completely lost, while vision in the right improved slightly.

Physical examination was negative.

Neurological examination was negative except for the ocular findings. There was swelling of both discs, more marked on the right. Fingers were perceived on the right at one foot, on the left at three inches.

There was too much of a local inflammatory reaction in the sinuses to allow of an evaluation of the existing condition. An x-ray showed clouding of the ethmoidal and antral cells. An x-ray of the sella turcica showed deepening of the sella posteriorly, with some thinning of the posterior clinoid process, but no erosion.

Two days after admission the ophthalmologist reported blurring of the discs bilaterally, with an elevation of three diopters. On the left disc there were a few fine hemorrhages. The spinal fluid was normal in all respects.

The diagnosis was bilateral optic neuritis, probably from diseased sinuses.

The patient was given typhoid vaccine. Five days after admission the sinuses were explored. The next day vision was nil in the left eye, while fingers were perceived at two feet with the right. A week later vision was 10/200 in the

left and 10/15 minus in the right eye. The disc margins were less blurred, the elevation being one diopter on the right, one-half diopter on the left. The hemorrhages had disappeared. A few days later vision in the right eye was 10/20 and in the left 10/30. At this time an exacerbation occurred. The patient complained of blurred vision in the right eye, vision in that eye was now 10/200, and a central scotoma was detected. This condition subsided in a few days, vision in the right eye being 10/30 and in the left 10/40.

The attending neurologist noted at this time definite pallor of both discs, and an inequality of the patellar reflexes. While the entire process, in his opinion, might have been due to infection, he was not certain that the infection could account for so great a degree of amaurosis and swelling of the discs.

The patient was subjected to a course of seven injections of neosalvarsan.

On October 6, a lowering of the condition was again noted. Vision in the right eye was 10/200 and in the left 8/200. The visual fields showed accompanying changes. Examination of the sinuses disclosed no suppurating foci. There was gradual improvement, vision becoming 10/15 in the right eye and 10/20 plus in the left. A neurologic examination showed hyperactive patellar reflexes, greater on the left, and a Hoffmann sign on the left.

Unfortunately the patient could not be observed after discharge from the hospital November, 1930.

Comment: In this case a bilateral optic neuritis was present at the onset, with definite evidence of disease in the sinuses. Following the sinus operation a see-saw type of fluctuation occurred, one eye becoming worse and the other better. Following a second exploration, the visual status improved, only to be followed by another exacerbation. Then followed improvement, another exacerbation and improvement.

The patient was in the age group for multiple sclerosis. The lack of any significant improvement after one operation, as well as the few but suggestive neurological signs of dissemination, may also be thought to favor the diagnosis of multiple sclerosis.

On the other hand, such a degree of optic neuritis, with hemorrhages in the disc, would be rather atypical for this condition. And it is conceivable that even if an optic neuritis is infectious in origin it need not, in every case, respond promptly to eradication of the focus if the changes set up in the optic nerve prove irreversible.

It is possible that this patient had multiple sclerosis, and an optic neuritis of infectious origin.

CASE 11.—M. O., a milliner, aged 39, entered the Mount Sinai Hospital, December 11, 1937, and was discharged January 23, 1938.

Past History: The patient had suffered frequent upper respiratory infections in the past, associated with severe frontal and occipital headaches.

Present Illness: Eight days before admission, the patient suddenly became aware of a large black spot on the nasal side of the field of vision of her left eye. This defect gradually increased in size up to the time of admission. A few days before the onset, the patient had had one of her usual upper respiratory infections; the infection had subsided, but the headaches persisted.

Physical examination was negative.

Neurological examination was negative, except for the ocular findings. There were bilateral corneal opacities (thought to be due to phlyctenular keratitis). The left pupil was slightly greater than the right and more sluggish in reaction to direct light; the consensual reaction of the left pupil was, however, adequate. Visual acuity on the left was diminished so that only moving fingers could be seen. There was a large central scotoma. There was perhaps slight peripapillary edema, more definite on the left. The ankle jerks were markedly diminished. The right knee jerk was possibly greater than the left. The abdominal reflexes appeared diminished.

The cerebrospinal fluid was under 96 mm. H_2O of pressure. There were four cells. The Pandy reaction was negative. The Wassermann reaction was negative. The colloidal gold curve was 2211110000. The total protein was 36 mgms./100 cc.

The serology of the blood was negative on two occasions.

On another occasion the left radial reflex was considered weaker than the right, the left triceps greater than the right, and all the abdominals except the left upper, absent.

An x-ray of the sinuses showed clouding of both antra, and of the anterior ethmoids.

The diagnosis considered at this time were multiple sclerosis and optic neuritis, secondary to sinusitis.

On December 15, a left spheno-ethmoidectomy was performed and gelatinous pus found in the posterior ethmoid cells.

Immediately after the operation vision became worse in the left eye.

The sinuses were irrigated and typhoid vaccine was administered. December 22 not even light was perceived in the left eye. A few days later, vision showed some signs of improvement. The patient was able to see fingers at four inches, a few days after that at eight inches; when last measured visual acuity in the left eye was 1/20. The fields showed accompanying changes with a breaking up of the scotoma. The left optic nerve appeared pale.

Neurological examination on two occasions subsequent to the last cited above failed to disclose any evidence of disseminated disease.

Comment: The final diagnosis in this case can only be made after further and long continued observation. Multiple sclerosis is still seriously to be considered in view of the suggestive, if transitory, positive neurologic signs, the abnormal colloidal gold curve and the apparent lack of relationship between the time of operation and the beginning of improvement. The other possibility is, in view of the onset following an upper respiratory infection and the obviously diseased state in which the sinuses were found, optic neuritis secondary to disease of the sinuses.

CASE 12.—N. S., a housewife, aged 36, entered the Mount Sinai Hospital, November 17, 1932, and was discharged December 9, 1932.

Past history was irrelevant.

Present Illness: One month ago vision in the left eye had become blurred, especially at the center of the field. The condition was most noticeable at night.

Physical examination was negative.

Neurological examination was negative, except for the ocular findings. Vision in the right eye was 15/10, in the left 15/70 plus. The fundi appeared negative.

Sinus examination disclosed evidence of a left sphenoiditis.

The blood, urine and cerebrospinal fluid were all negative.

It was decided that the patient required an immediate operation on her sinuses, and a left spheno-ethmoidectomy was performed. There was no improvement in vision, the visual acuity of the left eye becoming 20/200, and a scotoma appearing about the fixation point.

Because of the ineffectiveness of the operation the diagnosis of multiple sclerosis was now considered.

Unfortunately the patient did not present herself for further observation after discharge from the hospital.

Comment: If eradication of the infectious focus in the sinus is regarded as a specific therapeutic test then this case cannot be considered one of rhinogenic optic neuritis, but rather one of multiple sclerosis ushered in by retrobulbar neuritis, with merely coincidental involvement of the sinuses. The question here again arises, however, whether in every case of rhinogenic optic neuritis improvement need invariably follow eradication of the focus or whether an infectious focus in the sinus may not set off a similar process in the optic nerve, which, in some cases, proves refractory to therapeutic measures.

The failure of the patient to report further deprived us of the opportunity to establish a final diagnosis.

The following case is interesting as an example of an optic nerve complication from sinus disease of a type different from those we have thus far been discussing.

CASE 13.—B. G., a housewife, aged 29, entered the Mount Sinai Hospital, June 29, 1937, and was discharged July 15, 1937.

Past history was irrelevant except for the occurrence of frequent colds, and in 1934 an antral infection complicating an attack of influenza.

Present Illness: Six weeks before admission the patient had a hysterectomy for a fibromyoma of the uterus performed at another institution. A general anesthetic was employed (cyclopropane ethylene oxygen ether).

Three days after the operation the patient complained of dizziness and severe pain in the occipital and supraorbital regions and at the back of her neck. Cocainization of both sphenopalatine ganglions afforded no relief. Her vision became impaired, so that she could see only the outline of objects. She also developed tingling, numbness and pain in the middle fingers of both hands, and vomited frequently. A sinusitis was detected and treated with nasal sprays and irrigations. For a time a developing meningitis was feared.

Ophthalmologic examination by Dr. Kaufman Schlivek two weeks before admission to the Mount Sinai Hospital disclosed diplopia upon gazing in all directions. The visual fields appeared grossly normal. There was bilateral papilledema with hemorrhages and exudates. There was an elevation of three diopters in the right eye and two and one-half diopeters in the left. The ophthalmologist suspected an intracranial neoplasm.

The neurological examination, by Dr. Israel Strauss, disclosed glove and stocking sensory disturbances in the upper and lower limbs. There was a zone

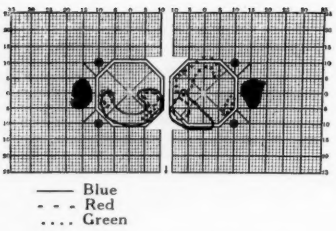

Fig. 13. Case 8. Four days before operation.

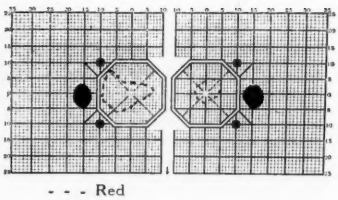

Fig. 14. Case 8. Eight days after operation.

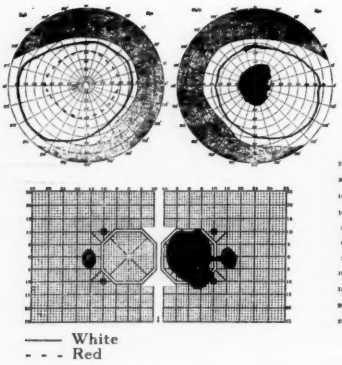

Fig. 15. Case 9. Status April 24, 1935.

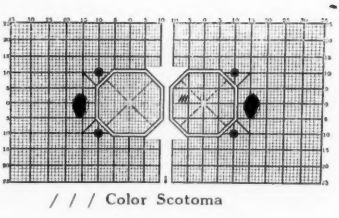

Fig. 16. Case 9. Status May 17, 1935.

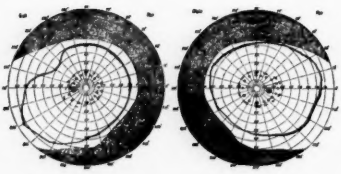

Fig. 17. Case 13. Seven days after operation.

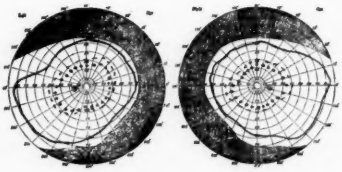

Fig. 18. Case 13. Twelve days after operation.

of hyperalgesia over the third and fourth cervical dermatomes bilaterally. The reflexes were markedly diminished, the knee jerks being almost absent. There appeared to be involvement of central vision. The neurologist did not consider the findings due to an expanding lesion, but more probably due to irritation by a toxic process, with involvement of the peripheral nerves and, perhaps, the spinal roots.

Ophthalmologic examination a week after the preceding examination showed progression. The elevation was one diopter greater in each eye; there were stellate figures in the macular region, and fresh hemorrhages.

Three days before admission to the Mount Sinai Hospital a lumbar puncture disclosed fluid under 500 mm. H_2O pressure. After 33.5 cc. of fluid were removed, the pressure was 150 mm. H_2O, yielding an Ayala index of ten.

On admission to the Mount Sinai Hospital, the patient felt better generally, but vision was unimproved. Examination of the sinuses showed the right middle turbinate moderately congested. There was a mucopurulent secretion in the right superior meatus, and mucopus in both sphenoid sinuses.

Ophthalmologic examination showed an elevation of four diopters in each eye with several fresh hemorrhages. There was edema about each macula, with a hemorrhage in the right macula.

The cerebrospinal fluid was under a pressure of 380 mm. H_2O. After removal of 10 cc. of fluid the pressure was 190. The Ayala index was five. There were two lymphocytes per cmm. The Pandy reaction was negative.

July 1, at the suggestion of Dr. Strauss, a bilateral spheno-ethmoidectomy was performed by Dr. Irving Goldman. Two days later the pressure of the spinal fluid was 120 mm. H_2O. 23 cc. of fluid were removed, after which the pressure was 60 mm. H_2O. The Ayala index was 11.5. There were 12 red blood cells per cmm. A few hours later the elevation of the right disc was found to be two diopters, that of the left one and one-half diopters. There were several fresh hemorrhages in both eyes and the changes in the macula appeared more marked. Diplopia was not present.

A week later the edema appeared more widespread in both eyes. There were fresh hemorrhages in the right eye, and the lesion in the left macula had increased. The elevation of the discs was unaltered. Visual acuity of both eyes was 15/30. (The visual fields are seen in the accompanying Fig. 17).

Eleven days after operation the fundus picture was much improved. The elevation of the right disc was one diopter, of the left one-half diopter. Both nerve heads were somewhat pale, the margin obliterated, but the peripapillary edema was less, as were the hemorrhages and exudates. The arteries were thin, and the veins engorged. The visual fields were likewise improved (see Fig. 18).

Ophthalmologic examination about four months after discharge from the hospital showed a vision of 15/20 in the right eye and 15/30 in the left; the right disc was pale, the margins serrated, the arteries narrow. There was marked perivasculitis about the nerve head. There was no elevation. In the macula there was an area of depigmentation. The left disc was pale; the arteries narrow and the margins of the disc serrated. There was marked perivasculitis about the nerve head. There were several hard white exudates in the macula. The fields of vision showed no change from the last examination. The findings were those of bilateral postneuritic optic atrophy.

Neurological examination at this time showed no abnormalities except for the ocular findings. The absence of an expanding lesion appeared confirmed, and the sinuses were considered responsible for her symptomatology. The patient continued to require treatment for her sinuses because of a chronic discharge.

Comment: The occurrence of dizziness, pain in the head, vomiting and the fundus findings of high-grade papilledema were well calculated to give rise to the suspicion of an expanding cerebral lesion. Although the clinical diagnosis of the pelvic condition was a benign tumor of the uterus, malignant degeneration with metastasis had to be reckoned with. However, the course was very fulminating, even for a metastatic process. There were, moreover, no focalizing signs. And if one predicated a periventricular growth to account for the absence of focalizing signs, some of the neurological findings pointing to involvement of peripheral structures would not be accounted for. In the face of undoubted evidence of increased intracranial pressure, the neurologist postulated some form of irritation from a toxic process. The Ayala index seemed subsequently to provide additional evidence against the presence of an expanding lesion. The high pressure of the cerebrospinal fluid and the absence of pleocytosis suggested a condition analogous to the otitic hydrocephalus of Symonds. Since there was a previous history of sinus disease, and there was found a definite suppurative process in the sinuses, they were tackled as a possible focus of infection. The end-result justified this method of attack. In retrospect it would appear that the infection of the sinuses liberated some toxins which were responsible both for the serous meningopathy and the peripheral neuritic phenomena.

This case, incidentally, lends strength to the argument that the term "otitic hydrocephalus" is a misnomer when used as a blanket term since infectious foci at other parts of the body besides the ear can produce a similar picture. If desired one might apply the term "sphenoiditic" hydrocephalus to this case.

DIFFERENTIAL DIAGNOSIS

The diagnosis of retrobulbar neuritis of rhinogenic origin rests, on the negative side, on the exclusion of all other possible causes for the neuritis, on the positive, on establishing a diseased state of the sinuses.

Foremost in the list of alternative causes is multiple sclerosis. The frequency of retrobulbar neuritis in this disease is variously cited by different investigators, depending no doubt on the length of time the cases under observation have had the disease. In younger individuals multiple sclerosis must especially be borne in mind. A history of transitory symptoms of a neurologic nature such as diplopia, sphincteric disorders, paresthesias or paralysis may provide a clue in individuals neurologically intact at the time of the examination. In other cases neurologic examination may disclose few but unequivocal signs, which, together with the evidence of retrobulbar neuritis, indicate dissemination of lesions. Pleocytosis or a positive colloidal gold curve in the cerebrospinal fluid may provide confirm-

atory evidence. When the syndrome of multiple sclerosis is fully developed no difficulties should arise, while in other cases years of observation may be necessary to aid in establishing the diagnosis. If the fundus changes are very marked, with definite elevation of the optic disc, multiple sclerosis is less likely, since such changes, while occurring in multiple sclerosis, are unusual.

Leber's hereditary optic atrophy is another condition which must be differentiated, producing as it does the syndrome of retrobulbar neuritis. The history of familial incidence is of great value in the differentiation. It is important to remember that variants from the typical form occur, that the disease may occasionally affect females as well as males, that there may be remissions in the course of the disease, that one eye may be affected for some time before the other. The onset of visual impairment is generally gradual so that there should be no difficulty in differentiating the condition from cases of rhinogenic neuritis with the usual rapid fulminating onset.

Neoplasms which arise in the vicinity of the optic nerve, such as subfrontal tumors or tumors in the chiasmal region may by pressure on the optic nerve produce visual disturbances similar to those of retrobulbar neuritis. Here, too, the onset is more gradual and other symptoms and signs characteristic of these conditions should prove of aid. Aerograms, or plain x-rays of the skull, taken with the special view to disclose abnormalities in the region of the optic foramen, may be necessary in certain cases.

Syphillis is not a frequent cause of retrobulbar neuritis. In this disease the pathologic process, when it affects the optic nerve, is primarily meningeal, in the sheath of the nerve, then spreading to affect the outermost fibers. The visual disturbance produced is a contraction of the peripheral visual field and not a central scotoma. In rare cases, however, syphilitic retrobulbar neuritis has been described. The measures necessary to differentiate the conditions are, of course, obvious.

Toxic retrobulbar neuritis, such as that caused by alcohol or nicotine, is bilateral in site and gradual in onset. A history of excessive indulgence provides diagnostic aid. In the case of tobacco sensitivity tests on the skin may prove of additional value.

Rarely, infectious diseases such as erysipelas, mumps, typhoid fever, pneumonia, malaria, scarlet fever, measles, chicken pox, smallpox and diphtheria produce retrobulbar neuritis.

In those cases in which the process attacking the optic nerve spreads forward to the nerve head, a picture of optic neuritis results with reddening of the disc, blurring of its margins, with an elevation of several diopters, hemorrhages and exudates. The appearance may be indistinguishable from that of papilledema or choked disc, due to increased intracranial pressure. In several of our cases the necessity of differentiating between the two conditions arose. Since the picture presented by the fundus may be identical in the two conditions additional factors must be relied upon: symptoms and signs of increased intracranial pressure, focalizing evidence of an expanding lesion, cerebrospinal fluid pressure measurements, aerograms of the brain. One differential diagnostic point of considerable value, as a rule, is that in optic neuritis the visual impairment is out of proportion to the changes in the fundus, while in papilledema, in the early stages at least, negligible visual disturbances occur.

DISCUSSION

Several theories have been proposed to explain the pathogenesis of rhinogenic optic neuritis. One holds that there is a spread of some noxious agent from sinus to optic nerve, either by direct extension, or via venous or lymphatic channels; another, that the optic nerve is affected by compression in the optic canal through venous stasis or from swelling of the wall of the canal; another, "reflex" theory maintains that the condition is brought about by spasm of branches of the ophthalmic artery in reflex response to irritation of trigeminal nerve branches in the sinus; finally, an allergic mechanism is invoked by some, the inference being that the optic nerve shows some sensitivity to bacterial proteins in the sinuses. In the present unsettled state of our knowledge it is impossible to decide which of these theories is the correct one. Since the condition in uncomplicated cases does not terminate fatally we are not in possession of pathologic studies which might throw light on this subject.

Certain radiographic studies reported by White[9] may have a bearing on the pathogenesis of the condition. Following the suggestion originally made by van der Hoeve, White studied the optic foramina of normal individuals and of individuals with presumptive rhinogenic optic neuritis. He found that in normal individuals the average horizontal and vertical diameters of the canal were 5.18 and 5.52 mm. In 25 cases with optic nerve involvement the diameters were 4.45 and 4.92 mm. In every unilateral case, the canal on the side involved was the smaller. When both eyes were affected

the smaller canal was on the worse side. He stated, "This seems to demonstrate that the smaller the canals the more serious the neuritis." "The smaller the canal the more the danger of permanent loss of vision and the greater the necessity for exploration. The size of the canals is most valuable in making a differential diagnosis, for large canals immediately lead one to look elsewhere than in the accessory sinuses for the cause of the amblyopia." If substantiated, the findings of White would provide an important clue to the mechanism of the production of rhinogenic retrobulbar neuritis. Actually some investigation along similar lines has been carried out. Goalwin,[10] in his study of the clinical value of optic canal roentgenograms seemed to confirm the conclusions of White. He stated, "Optic canals below the average size are the rule in cases of optic nerve involvement which is secondary to sinus infection and other focal infection. In cases of unilateral involvement it is the side with the smaller canal that is usually involved." However, Goalwin sets as the average diameters of the canal in normal individuals 4.3 and 4.4 mm., and as minimum diameters in normal individuals 3.25 and 3.5 mm. The latter, it will be noted, are lower than the figures found by White in cases of optic nerve involvement, rendering the criteria set up by White questionable at least. Pfeiffer[14] more recently has criticized previous techniques as inaccurate and has devised a new technique, wherein the head is fixed and the x-ray tube shifted, to overcome the sources of error. Obviously further studies along this line are needed. In our series of cases no systematic investigation of this phase of the problem was made.

Gradle[12] has formulated a stimulating conception of the mechanism of optic neuritis from disease of the sinuses. To understand his explanation it is necessary to review briefly the anatomy of the so-called vein of Vossius. From the posterior orbital portion of the optic nerve and its sheaths there arise small veins that gradually approach the center of the nerve in its canalicular portion, there uniting to form the posterior central vein of the optic nerve. This vein receives branches not only from the nerve and its sheaths, but also from the posterior aspects of the orbital periosteum. The vein leaves the nerve at the posterior end of the optic canal and empties into the cavernous sinus.

Gradle believes that the pathogenic agent from the sinuses by continuity reaches the intracanalicular portion of the dura of the optic nerve, or possibly through the periosteal or dural veins reaches the central vein of Vossius. If the dura alone is involved, causing pressure on the optic nerve within the canal, the peripapillary

bundles alone are involved and an enlargement of the blind spot ensues. If the process extends further and involves the central vein of Vossius the neighboring nerve bundles suffer. These happen to be the papillomacular bundles and there results a central scotoma.

No discussion of the pathogenesis of rhinogenic optic neuritis is complete without mention of the outstanding and oft-cited anatomical researches of Onodi,[13] who demonstrated by painstaking study that great variability exists in the size of the sinus cavities, in the thickness of the bony wall that separates the sinuses from the optic nerve and in the relationship of the posterior ethmoidal and sphenoidal sinuses to the optic nerve. Onodi demonstrated that there are subjects in whom the optic nerve is separated from the sinus by the thinnest layer of bone or by mucous membrane alone. A similar report dealing with the variability in the structure of the wall separating the mucous membrane of the sinus from the dural sheath of the optic nerve was made by Herzog.[14]

It may not be amiss at this point to present the following case, which demonstrates the susceptibility of the optic nerve to pathological states in the accesory nasal sinuses.

REPORT OF CASE

CASE 14.—A. N., a printer, aged 43, entered the Mount Sinai Hospital, January 25, 1937, and was discharged May 2, 1937.

Past History: The patient had had an operation on his sinuses about nine years ago and a bilateral antrotomy one year ago. For many years he had suffered supraorbital headaches attributed to his sinus condition.

He entered the hospital for relief of a sciatic syndrome, attributed to sacroiliac arthritis. He was treated with epidural injections of novocain, but since he was not benefited, and since his sinuses were found diseased, a right spheno-ethmo-antrotomy was performed. It was necessary at the time to pack the nose to control rather profuse bleeding from the region of the antrum and the sphenopalatine artery. A week later revision of the spheno-ethmoidectomy was performed. Profuse hemorrhage occurred in the sphenoid sinus, for which packing was again necessary. This was removed entirely three days later. Three days following the removal, the patient showed signs of retrobulbar neuritis. Vision in the left eye was 3/200. The fundus appeared normal, but there was a definite central scotoma.

The ophthalmologist considered the retrobulbar neuritis due to an inflammatory condition of the sphenoid sinus secondary to the packing that was inserted to halt the bleeding.

A large mucopurulent blood clot was removed from the sphenoid and the sinus was irrigated.

Progressive improvement followed this procedure. Vision was finally 20/20 minus 2 in the right eye, and 20/25 minus 1 in the left. The central scotoma cleared up rapidly.

The important practical problem of just what to do when confronted by cases of the type we have described rests, ultimately, on one's conception of the nature of the disease, on whether one predicates a causal relationship between disease of the sinuses and optic neuritis or not. As already mentioned, the sequence of suddenly appearing blindness, detection of diseased sinuses, surgical intervention, improvement, in the minds of many leaves no doubt about such a relationship. Others, however, reviewing the same body of facts, deny categorically any such relationship and hence consider surgery ill-advised. We propose now to examine various arguments cited by the opponents of the theory of rhinogenic optic neuritis, and the counter arguments of its protagonists.

One argument cited against the theory is that since sinusitis is such a common ailment and retrobulbar neuritis so rare it is difficult to conceive of a connection between them. This reasoning while seriously advanced by some does not bear serious scrutiny. It is possible to have rare as well as common complications of a disease entity. Cerebral abscess is not a very frequent complication of sinusitis, but no one doubts the etiologic relationship in that case. There doubtless are factors, as yet unknown, which determine why only in a small number of cases disease of the sinuses leads to involvement of the optic nerve. The anatomical variations described by Onodi may be one such factor. Some observers, it should be added, while granting that involvement of the optic nerve from disease of the sinuses rarely occurs to a degree sufficient to assume clinical proportions, maintain, nevertheless, that milder sub-clinical involvement is not uncommon. Thus Markbreiter reported visual field defects of various types in 70 per cent of individuals suffering from sinusitis. Bordley found enlargement of the blind spot in 31 of 102 such cases. Van der Hoeve reported changes in the visual field in practically all cases of posterior sinusitis. Admittedly, such findings are not universally accepted. Thus, Herzog was able to detect abnormalities in only 2.6 per cent of the subjects investigated.[15]

Reasoning similar to the above has been employed against the contention that since sinusitis is so frequent in children retrobulbar neuritis should be more prevalent at that age. It has been pointed out that the optic canal in children after the age of 3 years is as large as in adults and that the optic nerve, therefore, occupies a relatively more spacious canal, rendering it less vulnerable to the effects of pressure in that portion.

Behr,[5] who denies the existence of such entity as rhinogenic retrobulbar neuritis, cites, among other objections, the fact that if

an infectious process were to spread from the sinuses to the optic nerve the peripheral fibers of the nerve should be first involved, yielding defects in the peripheral field of vision. Two objections to his conclusion suggest themselves: first, we are not certain that the mechanism in rhinogenic optic neuritis is that of direct extension producing involvement of the superficial fibers before other fibers are involved; second, even in conditions where one *a priori* would expect the peripheral fibers first affected, as in pressure from tumors, the earliest evidence of involvement, supposedly because of the greater vulnerability of the macular fibers, may be a central scotoma.

It is contended that improvement of the optic neuritis after operation does not prove that the sinuses were responsible for the neuritis. This argument, of course, warns against a post ergo propter type of reasoning. Here one may distinguish two lines of contention. One maintains that whatever improvement occurs following the operation would have occurred if no operation had been performed; the other that the improvement is due to certain incidental procedures during the course of the operation other than the eradication of an infectious focus. The strongest advocate of the former view is v. Hippel.[6, 16]

Analysis of v. Hippel's report discloses the fact that his estimate of improvement in 70 per cent of cases operated upon is really an unnecessarily modest one, at least from his point of view. For, if following his lead, one excludes those cases that could not possibly have benefited from any form of therapy and includes only those which were amenable to therapy, then he obtained satisfactory results without surgery in 100 per cent of his cases! What v. Hippel claims in essence is that all cases potentially able to improve will do so without operative intervention, that those cases improving after operation do so in spite of and not because of this therapy. The question arises, however, whether the unfavorable cases in his series were all really as hopeless for all forms of therapy as he assumed. His failure to find disease of the sinuses in any of the cases is so at variance with those of other investigators that there is room for doubt about other of his criteria and the conclusions based thereon. There remains the possibility, at least, that some of his unfavorable cases might have fared better if less passive therapy had been resorted to.

It may be added that because certain of these cases recover without operation one should not conclude that the optic neuritis is not of rhinogenic origin. The inflammatory process may subside without benefit of surgery. To conclude as v. Hippel does that because cases improve spontaneously they cannot be due to an

infectious process in the sinuses is no more warranted than the reasoning v. Hippel takes exception to, namely, that because cases recover after operation the sinuses must have been responsible. Spontaneous cures may be used as an argument against the necessity of surgical intervention, not against the rhinogenic origin of optic neuritis.

Another line of reasoning runs to the effect that whatever beneficial results follow upon operation are due not to the eradication of a focus of infection, but to measures incidental to the operation. In other words, a causal relationship between disease of the sinuses and optic neuritis is not admitted, but it is granted that the operative procedure inadvertently has a beneficial effect. Reference is made to those cases in which nothing or relatively little of a pathologic nature is found in the sinuses and yet improvement follows operation. Thus Benedict[4] believes that the cures thus obtained may be due to the cocain-epinephrine anesthetic, which produces ischemia and then congestion in the vessels of the optic nerve, and to the absorption of blood following the operative trauma, which leads to a rise in temperature, these two factors producing a state of affairs similar to that induced by injection of foreign protein. Other observers point to cures following simple adrenalization or cocainization of the nose. Of course if such a relatively benign procedure did yield results comparable to those obtained after surgery it would be the method of choice. At the present time this has not been firmly established, however.

Our own attitude, after considering all these views, may be formulated as follows:

Mindful of the frequency of multiple sclerosis as a cause of retrobulbar neuritis, of the large percentage of cases of retrobulbar neuritis that spontaneously recover as well as of the cases that are reported cured after simple adrenalization of the nose, mindful of the fact that diseased sinuses are present in such a large proportion of our population that they may erroneously be incriminated as the cause of a host of conditions, and mindful also of the occasional untoward effects of nasal surgery, it is nonetheless our opinion that in some cases operative intervention is indicated. We do not go to the extreme, advocated by some, of insisting on histologic examination of tissue before dismissing the sinuses as normal. But if the rhinologist, specially trained, concludes as a result of his examination that the sinuses are definitely diseased, and no other cause for the neuritis is apparent, we favor eradication of the focus. Especially

if continued observation shows progression in the visual impairment. It may be that a number of our cases would have improved spontaneously. It may be that some of our cases will eventually turn out to be instances of multiple sclerosis. But no one can be certain that other of these cases, if left untreated, would not have developed permanent optic atrophy. And who, so long as such a possibility exists, will be content to wait a decade or more on the chance of multiple sclerosis finally announcing itself instead of taking remedial measures at once? A healthy reaction away from indiscriminate nasal surgery should not lead one to the other extreme of withholding surgery when it seems urgently indicated; especially since, in competent hands, untoward effects are rare. Even in those cases in which the diagnosis of multiple sclerosis is well established, if a retrobulbar or an optic neuritis is present and diseased sinuses, which may be the responsible factor, are found, we think it justified to treat the sinuses just as if multiple sclerosis did not exist.

As an adjuvant in therapy we have found it useful to employ foreign protein in the form of typhoid vaccine. The hyperthermia artificially induced appears beneficial in hastening the process of recovery.

116 West 59th Street.
70 East 83rd Street.

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LXXXII

OTOGENOUS CEREBELLAR ABSCESS*

MILES ATKINSON, M.D., F.R.C.S. (ENG.)

NEW YORK

Abscess in the cerebellum being a rather uncommon occurrence, it is accorded in the literature less notice than abscess above the tentorium and perhaps less notice than is its due. It has its own not inconsiderable problems of diagnosis and treatment quite distinct from those of cerebral abscess, and its mortality is appreciably higher, possibly because of this lack of notice. It does not seem inappropriate, therefore, to bring forward again some of the more important considerations in its regard.

To speak of otogenous cerebellar abscess is almost tautologous, since sub-tentorial abscess is almost invariably secondary to suppuration in the ear. Single metastatic abscess is so uncommon that it needs no more than mention. By chance I have had one such case which was secondary to an osteomyelitis of the tibia.[2]

Infection may pass from the ear to the cerebellum by any one of four routes—the lateral sinus, the labyrinth, the triangle of bone between the two (Trautmann's triangle), or rarely directly along the Fallopian aqueduct.

I. *Lateral Sinus:* When the lateral sinus groove is involved one of two things happens—either (1) a perisinus abscess is formed which causes a localized meningitis, and infection enters the cerebellum by a perivascular path; or (2) the vessel itself is directly involved, becomes thrombosed, and infection extends inwards as a retrograde thrombophlebitis.

II. *Labyrinth:* In view of the many possible pathways through the labyrinth, it is surprising that labyrinthogenic abscess is not more common.

A. Preformed Paths: There are four. The internal auditory meatus and the aqueduct of the cochlea are both large canals which

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communicate directly with the sub-arachnoid space. Although they can and sometimess do localize infection—as witness "Politzer's abscess" in the internal auditory meatus—they offer relatively little resistance to the passage of infection, and infection along one of these paths is more likely to advance unopposed to the sub-arachnoid space and produce a generalized meningitis.

The aqueduct of the vestibule, on the other hand, is a canal with a narrow lumen very apt for the formation of adhesions. It is the most common pathway from labyrinth to cerebellum. Usually the infection proceeds by erosion of the bony wall of the aqueduct and forms an extradural abscess in the posterior fossa. But the canal contains the ductus endolymphaticus, and it is possible for infection to pass directly along this (Friesner and Braun[7]), and cause an abscess in the blind end (empyema of the saccus), a localized meningitis and so invade the cerebellum. Such a course is in the nature of a rarity. Fraser,[5] in analyzing 100 cases of suppurative otitis media which died of intracranial complications, found involvement of the labyrinth in 35 cases, and in only one of these a saccus empyema.

The hiatus subarcuatus is very occasionally the route taken by the infection (Fremel[6]). It contains a minute process of dura which can in some cases be followed into the mastoid antrum.

B. Bone Necrosis: (a) Infection can invade the bony wall of the aqueduct of the vestibule as has been stated.

(b) Involvement of the deep perilabyrinthine cells may cause an extradural abscess which, by extension outward and inward, can produce at the same time both a labyrinthitis and a cerebellar abscess (Fig. 1).

(c) Finally, the infection causing a labyrinthitis can spread into the internal auditory meatus and produce thrombosis of the internal auditory artery, when the entire bony labyrinth will be cast off as a sequestrum, as happened in a case of my own. The suppuration around this spreads to the dura and through to the cerebellum.

III. *Trautmann's Triangle:* The area of bone between labyrinth and sinus, is the commonest site for the passage of infection, whether the aural suppuration be acute or chronic.

IV. *The Fallopian Aqueduct:* This path is mentioned by Friesner and Braun,[8] who admit its rarity. The infection invades

Fig. 1. Temporal bone from case of cerebellar abscess showing coincident involvement of labyrinth and lateral sinus.

the bony aqueduct, involves the facial nerve, and then travels backwards along the course of the nerve to the cerebellum. Though rare, this route of infection is worth remembering. The initial facial paresis, appearing in the course of a chronic suppurative otitis media or following a mastoid operation, and with at first no signs of intracranial complication, can be very deceptive. I have seen—and failed at first to recognize—one such case.[2]

SITE OF ENTRY

If the sites of these various apertures and of the lateral sinus are mapped out on the surface of the cerebellum (Fig. 2), it will be seen that the internal auditory meatus, the aqueduct of the vestibule and the knee of the sinus all lie on or immediately below the great longitudinal fissure, while Trautmann's triangle and part of the sinus close to the knee lie on the postero-inferior lobule. This lobule, then, should be the one most commonly involved by an abscess, and this is found to be the case. The point is one of importance when exploration comes to be undertaken.

Having reached the meninges, the infection produces a localized meningitis and across this bridge it passes to the cerebellum. As a result of microscopical investigations of a large number of specimens I have found[1] that infection enters usually along the perivascular

space surrounding each cortical vessel, less commonly along a vascular path by retrograde thrombosis. The opposite view is held by Eagleton,[4] but he offers little real evidence for his belief.

To understand the invasion correctly, it is necessary to recall briefly the anatomy of the cerebral vessels. The cerebrum is supplied by two sets of vessels—central vessels which come up from the base of the brain, supply the white matter and the central nuclei and finally turn out towards the cortex, a short distance beneath which they terminate as end arteries; and cortical vessels which, passing outward over the surface of the brain, supply the grey matter of the cortex by a large number of small vessels, also end arteries. Between the two—central and cortical—is a zone of white matter lying immediately beneath the cortex, which is relatively poorly supplied with blood, and which I have called the "avascular zone." In the cerebellum, by reason of its greater convolution, this zone comes to occupy the center of a folium (Fig. 3), and it is in this area of poor blood supply, this avascular zone, that the abscess starts (Fig. 4).

Furthermore, it is found that one folium and one folium only is involved and that the abscess remains confined to that particular one (Fig. 5). In cases of vascular origin it sometimes happens that more than one vessel is thrombosed, so that more than one abscess may be formed. Even in such a case the tendency is for each abscess to remain confined to its own folium and for coalescence to take place late if at all.

Extension takes place inward as far as the base of the folium, where it is held up by the relatively well-supplied central mass of white matter, but mainly backward through the folium in which it started, pushing away the folia on either side as it increases in bulk, but never overstepping the limits of its own (Fig. 5). A cerebellar abscess thus tends to remain cortical, and to involve the central mass, and hence the central nuclei, mainly by edema and very little by suppuration. This is an important point in connection with symptomatology.

It follows from what has just been said that the abscess is ovoid in shape, the long axis from before backward. Moreover, if the virulence of the causative organism is sufficiently low for localization of infection to take place, a definite limiting membrane will be formed which has smooth walls (Fig 5). These statements are contrary to the generally accepted opinion. Fremel[6, 7] says that the

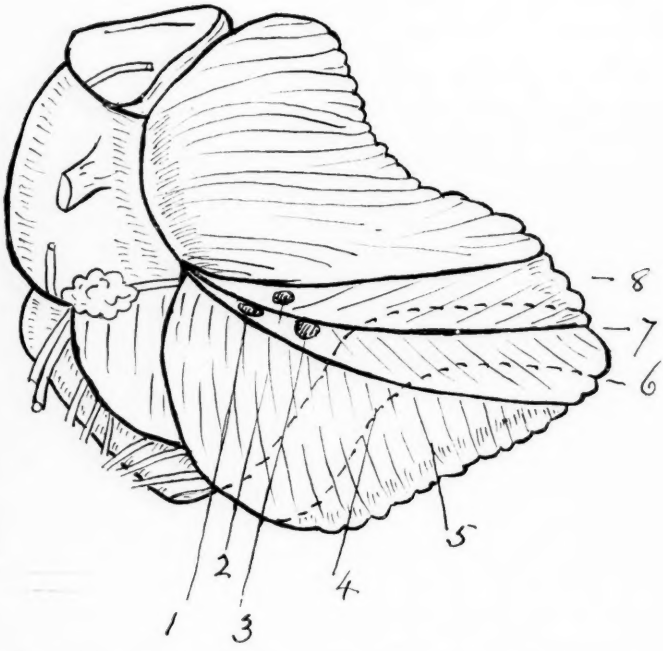

Fig. 2. Diagram (modified from Friesner and Braun) to show relation of surrounding structures to the cerebellum.

1. Internal auditory meatus.
2. Hiatus subarcuatus.
3. Saccus endolymphaticus.
4. Lateral sinus.
5. Biventral lobule.
6. Postero-inferior lobule.
7. Great horizontal fissure.
8. Postero-superior lobule.

abscess starts between central white matter and cortex, which latter it strips off like the peel of an orange, and that it grows in, and increases in size at the expense of, the central mass. He further states that in his experience one of the great characteristics of cerebellar abscesses is their irregular shape, and their tendency to extend upward into the vermis, pushing out fingers of infection in various directions on the way. This, he holds, largely accounts for the difficulty in draining them and for the high mortality. I have examined 17 specimens by serial section macroscopically and microscopically and have been unable to confirm his findings.[1] I do not

believe that the alleged widespread and irregular infection occurs at all commonly, or that it is the essential factor in the high mortality rate.

Acute infection gives rise only to some 20% of all cases of cerebellar abscess. In them it is generally agreed that the path of entry is by way of the sinus or the triangle in almost every case and in about equal proportion. Rarely is it by way of the labyrinth, the usual intracranial sequel to acute labyrinthitis being meningitis.

Chronic infection is responsible for the remaining 80% of cases, but there is not the same unanimity of opinion as to the route of entry. The Viennese School under Neumann[11]—I speak of the days before the Degradation—and indeed the Continental Schools in general (Lund[10]) believe that entry is by the labyrinth in about 50% of cases, rather less often by the sinus and seldom by the triangle. In England, on the other hand, labyrinthitis has not been found to be the prime offender. Turner and Fraser[12] in Edinburgh found cerebellar abscess as a complication of suppurative labyrinthitis resulting from chronic middle ear suppuration in only three out of 26 cases. In my own series of 14 cerebellar abscesses, only three resulted from labyrinthitis, while five resulted from sinus involvement, six from bone necrosis in the triangle.

The pathology may be summed up in a few words, that infection passes to the cerebellum more commonly as a result of chronic than of acute aural suppuration; usually by way either of lateral sinus or Trautmann's triangle and seldom via the labyrinth; and that it tends to remain cortical and confined to one folium.

SYMPTOMATOLOGY

The classical picture of unilateral cerebellar disease is too well known to need description here, but there are one or two points having special reference to abscess which must be mentioned.

1. The general signs of a space-occupying lesion within the skull are those of increased pressure. When the lesion is in the posterior fossa these signs are more marked than when it is supratentorial because pressure on the iter produces some degree of internal hydrocephalus, and when the lesion is an abscess they tend to be rapid in onset and severe in degree. They may therefore be of assistance in differentiating a cerebellar from a cerebral abscess when focal signs are few. Headache appears early, is more persistent and severe than in cerebral cases, and is often localized in the occipital region. Vomiting tends to replace nausea, and

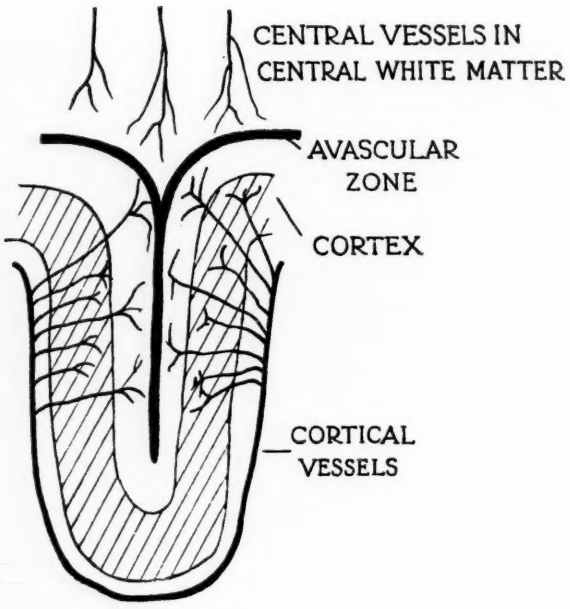

Fig. 3. Diagram of avascular zone in cerebellum.

papilledema is more frequent and of higher grade—one of my cases had five diopters of swelling. Wasting is often rapid and considerable, and may then be a sign of localizing value in a difficult case. Meningeal signs are not uncommon and sometimes cause difficulty in diagnosis.

2. The focal signs are equally familiar, but one or two points are worth emphasis. Cerebellar nystagmus is slow, coarse and horizontal, in contrast to the rapid, fine, rotary nystagmus of labyrinthine disease, and is characteristically to the side of the lesion, though it may be accompanied by a rapid, fine, irregular nystagmus to the opposite side which may cause confusion; falling is to the affected side; pointing error is always outwards and with the homolateral arm only; ataxia and dysdiadochokinesis are usually present in some degree, especially the former, and complete a clinical picture which cannot be mistaken when present in fully developed form.

"Silent" Abscess: Unfortunately this characteristic symptom-complex is not always produced. The cerebellar cortex is a silent area. Bárány's theories of cortical localization in the cerebellum have been disproved by Gordon Holmes' investigation of war wounds, by Magnus and de Kleijn's animal experiments on cerebellar extirpation, and by the results of partial resection of the cerebellum in man, and it is now generally accepted that the classical signs of cerebellar disease are produced only when the deep nuclei, in particular the dentate, are involved. This is frequently not the case with cerebellar abscesses, which are cortical and well away from the central nuclei. The interval may be bridged by the surrounding encephalitis, but not always (Fig. 5), and it is in this way quite possible to have a cortical cerebellar abscess which presents no cerebellar signs. I have on previous occasions[1, 2] urged the importance of these "silent" abscesses both in cerebrum and cerebellum, and pointed out that they are far from uncommon. Four out of my 12 cases were of this type, two of them found unexpectedly at postmortem, while the other two showed no focal signs until operation had stirred up the surrounding inflammation.

Thus, while a cerebellar lesion with full-fledged focal signs makes an unmistakable picture, it is possible, indeed it is not infrequent, to find an abscess in the cerebellum which is entirely devoid of them.

3. Paralysis of the Medullary Centers: It may not be out of place to issue once again a warning of the danger of lumbar puncture in posterior fossa cases. The danger is admittedly greater in tumor cases than in abscess, but even in the latter lumbar puncture is not to be undertaken light-heartedly and the very minimum of fluid should be drawn off for diagnosis only. What happens is that, as fluid is removed from below, the pressure above forces the medulla down into the foramen magnum producing a so-called medullary cone, and respiration ceases from paralysis of the center. It is sometimes possible, by immediate ventricle tap and drainage of the abscess while artificial respiration is being maintained, to get the center working again, but ultimate recovery after this accident is uncommon. This warning is constantly being reiterated and almost as constantly being ignored, so that unnecessary fatalities continue to occur.

DIFFERENTIAL DIAGNOSIS

There are five conditions from which cerebellar abscess has to be differentiated.

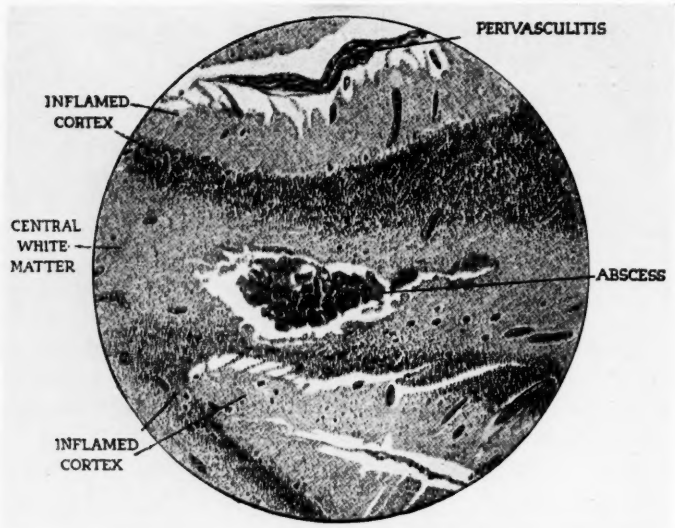

Fig. 4. Abscess in center of white matter of a lobule.

1. Acute Suppurative Labyrinthitis: The difficulty here is not only that the signs of the two conditions are very similar, but that both may occur in one and the same patient at one and the same time. Labyrinthitis, extradural abscess and cerebellar abscess form a well-recognized triad. The points of greatest differentiating value are as follows:

(a) Nystagmus: In labyrinthitis the direction of the nystagmus is never to the side of the lesion after the first few hours, and after the acute onset it tends progressively to subside. In abscess, the direction is variable but characteristically to the side of the lesion, and it may change in direction and intensity from day to day.

(b) Falling: In labyrinthitis, the direction of fall is always in the direction of the slow component of the nystagmus and alters with the position of the head. In abscess, it is always to the side of the lesion regardless of the direction of the nystagmus or the position of the head.

(c) Pointing Error: In labyrinthitis, the pointing deviation affects both arms and is always to the side of the slow component of the nystagmus. In abscess the deviation affects the homolateral arm only and is always outward.

When both conditions, labyrinthitis and abscess, are present at the same time, the diagnosis can be exceedingly difficult. Elucidation of the problem will depend upon a careful analysis of the three signs discussed above. It may even be that complete diagnosis will only be possible by ablation of the labyrinth, which, by removing all signs due to disease in that organ, leaves the underlying cerebellar picture revealed as on a palimpsest.

2. Frontal Lobe Abscess: To mistake the signs of a frontal lobe lesion on one side for those of a cerebellar lesion on the other is not difficult. Although it has been called the house-surgeon's mistake, it is not unknown among those of much greater experience. Frontal headache, nystagmus and vertigo are signs common to each, the aphasia of the one may be confused with the slurred speech of the other, and paresis of one arm may be present in either condition. The alert observer alive to the difficulty will, however, find other signs which will enable a correct diagnosis to be made.

3. Cystic Serous Meningitis: This condition has been fully described by Jenkins.[9] It is characterized by a history of previous ear infection, the signs of a posterior fossa lesion with vertigo as the marked presenting feature, and a normal spinal fluid under considerably increased pressure. In chronic cases the condition is usually of too long standing for any suspicion of an abscess to arise, but when it follows closely on acute disease of the ear, as in a case of my own, the simulation can be remarkably close. Jenkins advised repeated examination of the spinal fluid to provide the clue to diagnosis—dangerous advice as has already been pointed out. Exploration will be needed in any event and should be undertaken early.

4. Tuberculous Meningitis: This may sound far-fetched, but the signs can be unexpectedly similar. It is possible, indeed not uncommon, to have a cerebellar case in which meningeal signs are prominent and focal signs absent. In each disease the onset may be insidious or acute, the temperature and pulse low, meningeal signs present. The spinal fluid picture may be confusing, for in both there is an increase of pressure and cells, and the latter being mainly lymphocytes will fit in with either. Even the finding of a dead labyrinth does not solve the problem, for this might be

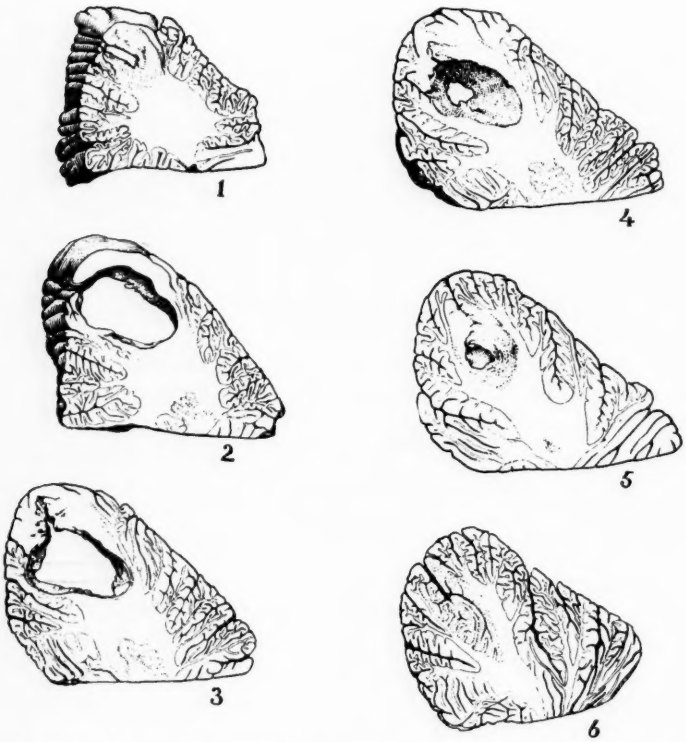

Fig. 5. Cerebellum showing unilobular involvement, also regularity of outline of the cavity, and wide separation from the dentate nucleus.

due to tuberculous disease and be the cause of the meningitis. The diagnosis may ultimately have to wait upon the appearance of focal signs or the fate of a guinea pig.

5. Tumor is always mentioned here, but rarely presents serious difficulty. The aural infection must always make one very suspicious of abscess. If the abscess is of so chronic a nature as seriously to simulate a tumor, differentiation is of small importance, for it could be treated as a tumor and excised in toto. I do not know of a case in which this procedure has been practiced in the cerebellum, though it has been done many times in the cerebrum.

TREATMENT

There is no generally accepted method of procedure, and Brunner[3] in 1936 went so far as to say that operative technique was "still experimental," too broad a statement but indicating the lack of unanimity in this matter. In general terms, it may be said that there are two methods of approach, one through the mastoid following the track of the infection, the method most favored by otologists; and the other through a clean area behind the lateral sinus, the method of the neuro-surgeon. The choice of route is conditioned probably more by the fact that each feels happier in his own field than by any great superiority of one approach over the other. Both have their merits—the mastoid route in that the source of the infection is cleared and the site of the abscess is close at hand; the posterior route in that there is more room and a clean field. This question of a clean field is a point of which I think too much has been made, for it is seldom that the subarachnoid space behind the mastoid in these cases of intracranial extension has not been sealed off. In any event, it always can be. Discussion will probably best be served if I outline my personal preferences and my reasons.

Approach: A cerebellar abscess will be found in one of three situations, depending upon the route of entry of the infection (Fig. 6), and the approach will differ somewhat, according to which of the three positions it occupies. It is therefore important to make a careful preoperative study of each case from this point of view.

1. Behind the Labyrinth (Labyrinthogenic): A dead labyrinth provides the clue to these cases, which are the most difficult of any to treat. Moreover, they do not brook of much delay, for by their situation they soon exercise pressure on the brain stem. Luckily their situation means also that they will give early focal signs, lying as they do close to the anterior end of the dentate nucleus. The abscess is always small, closely applied to the posterior wall of the petrous pyramid and deeply situated, and can be missed with ease and drained only with difficulty by the "clean" approach. The only satisfactory approach is by way of the mastoid, doing a labyrinthectomy by Neumann's method from behind and exposing first the posterior semicircular canal. This gives direct access to the abscess and ample room for drainage since the labyrinth does not have to be preserved.

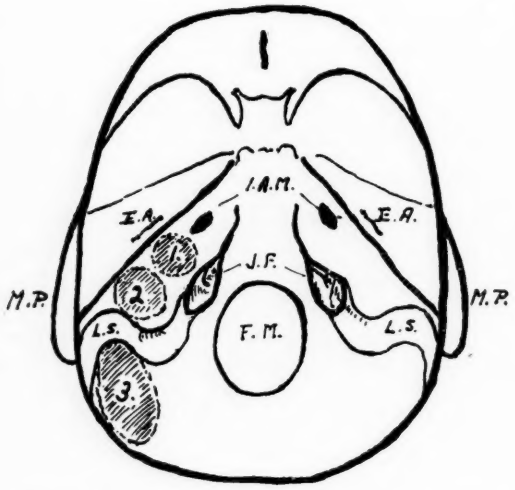

Fig. 6. Diagram showing sites of cerebellar abscess. 1. From labyrinth. 2. From Trautmann's triangle. 3. From lateral sinus.

I. A. M.—Internal auditory meatus. *F. M.* —Foramen magnum.
E. A. —Eminentia arcuata. *L. S.* —Lateral sinus.
J. F. —Jugular foramen. *M. P.* —Mastoid process.

2. Internal to the Descending Portion of the Lateral Sinus (Trautmann's Triangle): In this position the abscess is larger, more superficial and easier of access. Although it can be fairly readily approached by the "clean" route, it can be far more readily approached by the "dirty," and if the same precautions are taken to prevent spread of infection to the meninges in each case, I do not believe that there is any more risk of a meningitis being produced from without inward by the surgeon than there is from within outward by the abscess. I have not lost a case from this accident. The other objection, lack of room for drainage because one is confined by the labyrinth on one side and the lateral sinus on the other, is easily overcome by deliberate obliteration of the sinus and extension of the dural incision through the posterior wall, a maneuver which I have long advocated.[1]

3. Posterior to the Descending Portion of the Sinus (Sinus Origin): These cases, the most satisfactory of the three to deal

with when they are single, can be attacked equally well from their anterior end through the posterior wall of the obliterated sinus or from their posterior end through a clean approach.

Ventricle Tap: This, the invariable preliminary to operation on a cerebellar tumor, is just as important with an abscess. One of the great dangers in these cases is that of the pressure above forcing the medulla into the foramen magnum. A burr hole through which the lateral ventricle can be tapped is a safety valve through which supra-tentorial pressure can be regulated before, during and after operation. The high mortality in cerebellar as compared with cerebral abscess is, I believe, in large part due to lack of appreciation of the necessity for this precaution, even among neuro-surgeons who would not dream of operating on a sub-tentorial tumor without it.

Direction of Exploration: Because, as has been shown, these abscesses lie in the cortex, the direction of exploration when undertaken from the mastoid must be backward rather than inward or a small abscess may be missed. This, however, does not apply to those of labyrinth origin, which lie surprisingly far inward and forward because of the forward inclination of the petrous pyramid.

Drainage: Two rubber tubes are used, one large for drainage and one of half this diameter through which Ringer's solution can be run for irrigation. The smaller one should lie to the inner side of the larger so that the whole cavity is cleansed, and the relative sizes are important so that irrigating fluid shall not be dammed back.

Using these methods which I have outlined, I have had five recoveries out of eight operated cases, results which compare favorably with the average mortality of 75% in cerebellar abscess.

SUMMARY

1. An account has been given of the pathogenesis of cerebellar abscess, emphasizing its situation in the cortex, its confinement to one lobule and its regular shape.

2. The pathways of infection through the bone are described, and the frequency of labyrinthitis as a cause is discussed.

3. The classical symptomatology is briefly described and the fact of the existence of "silent" cerebellar abscess without the usual localizing signs is stressed.

4. Differential diagnosis from five conditions presenting similar signs is discussed, with especial reference to acute suppurative labyrinthitis.

5. Treatment is considered particularly from the point of view of the best route of approach according to the position of the abscess.

123 EAST 61ST STREET.

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LXXXIII

PROLONGED ANALGESIA AFTER TONSILLECTOMY BY NERVE BLOCKING ANESTHESIA

CAESAR HIRSCH, M.D.

NEW YORK, N. Y.

The agents ordinarily used for local anesthesia such as cocain, procain, tutocain, pontocain and nupercain have sufficiently long lasting anesthetic effects in surgery of the head and neck. If, however, we operate upon the tonsil, wounds are created in the direct path of the intake of food and swallowing is painful and difficult. Great benefit would be derived by the patient in the form of more rapid healing and less painful convalescence if long lasting postoperative comfort could be provided.

Many investigators have attempted to prolong the analgesia following local anesthesia in tonsillectomy. In 1914 Herzig[1] reported about 390 cases in which he operated upon tonsils and adenoids using urea and quinin hydrochloride. He injected a four per cent solution, made up with one per cent boric acid, into the anterior pillar of the fauces and applied a 50 per cent solution postnasally in the adenoid space. The analgesia in the pharynx lasted from two to eight days, the edema from two to ten days. The membrane formed at the site of the injection disappeared in from two to ten days. The author emphasized the advantage of urea and quinin over cocain.

In 1923 Sonnenschein[2] published his method of using a one-half to one per cent novocain or apothesin solution in a 33 to 50 per cent alcohol solution. The solutions were prepared just before their use, and ten drops of a 1/1000 epinephrin solution were added. Sonnenschein injected his solution following a procedure devised by Yankauer[3] in 1909 for blocking the posterior palatine nerve and combined it with the method described by John A. Thompson[4, 5] of injecting the glossopharyngeal tonsillar branches laterally from the tonsil. Sonnenschein claimed that he had excellent results with this method, and the patients seemed to have less discomfort than is usually noted in the first 24 to 72 hours following operation. However, several unpleasant features were noted. First, there was more pain at the time of injection than was the case when novocain or apothesin solution

was used. Furthermore, an impaired motility of the soft palate was observed which lasted for some time. This was due to blocking of the motor nerve. Sonnenschein modified his procedure later on by injecting an anesthetic solution containing from 25 to 33 per cent of alcohol into the region of the glossopharyngeal nerve, but using only procain or apothesin solution, without the addition of alcohol, for the blocking of the posterior palatine nerve.

Morgenroth[6, 7] and his co-workers during their studies of chemotherapeutic medications against trypanosomiasis, malaria, and diphtheria, found that various derivatives of quinin had not only a high germicidal but also an anesthetic power or, to use Ehrlich's terminology, there seems to be a peculiar connection between the parasitotrope and neurotrope function.

Eucupin base (isoamylhydrocupreine) ($C_{24}H_{34}O_2N_2$) (Fig. 1) is an almost tasteless white powder, insoluble in water and benzine, easily soluble in alcohol, ether and chloroform. Eucupin dihydrochloride ($C_{24}H_{34}O_2N_2$ 2HCL-H_2O) is soluble in 15 parts of water and easily soluble in alcohol. Eucupin was found to destroy many organisms, particularly the pyogenic cocci, with an efficiency reported to be 40 times that of phenol. This figure, however, is not a phenol coefficient since a time element is not involved and the antiseptic and disinfecting power of eucupin increases with the decrease in hydrogen ion concentration. For staphylococcus aureus its antiseptic dilution at pH6 is 1/10000, and at pH8 1/50000.

The local anesthetic action of eucupin is very marked. According to Morgenroth and Ginsberg the effect is from 20 to 25 times stronger than that of cocain. After an instillation of a 20 per cent solution of isoamylhydrocupreine anesthesia was observed which was still an absolute one after ten days, was somewhat diminished after 13 days, and only faded away after 16 days. A corneal opacity after the use of this 20 per cent solution was observed as an undesired by-effect. However, 20 per cent solutions have only been used theoretically in the laboratory since solutions of 0.125 per cent are efficient enough. De Takats[8] suggested eucupin with a combination of epinephrine and one of the more rapidly acting local anesthetics of short duration, and claimed that this combination produced a local anesthesia lasting for about 24 hours, without tissue reaction. Kilbourne[9] used eucupin 1/1000 concentration with procaine in proctologic surgery with good results and claimed that the anesthesia was unusually prolonged. The toxicity of eucupin is relatively low and is one-sixth that of cocain and but slightly greater than procain by

subcutaneous injection. The minimal lethal dose for mice is about 300 mgms. per kg. body weight. Intravenously, however, the toxicity for animals is higher and ranges from 30 to 70 mgms. per kg. body weight, depending upon the concentration of the solution injected; the latter figure applies to a 1/1000 dilution. These figures show that when eucupin dihydrochloride is employed as an infiltration anesthetic, toxic reactions are unlikely to occur.

Dawson and Garbade[10, 11] of the University of Texas carried out some investigations regarding the idiosyncrasy to various quinin derivatives. They stated that reactions to eucupin were not observed. In a later report, however, this statement was modified to the effect that such reactions were rare. These reactions soon clear up without any treatment. Fairly large doses of eucupin stimulate the vagus center and the respiratory center in the medulla. Perfusion experiments and irrigation experiments on smooth muscles have shown that eucupin is mainly inhibitory and causes a relaxation. The pressure effect of epinephrine is diminished by eucupin and consequently, when used as a local anesthetic, it is customary to employ more epinephrine as is the case when certain other local anesthetics are being used. The absorption of eucupin upon hypodermic or intramuscular injection is very slow. Its prolonged action is expressed by the fact that it remains in the tissues locally and may be recovered, according to Bylsma[12] for analysis four days after it has been injected. Within 24 hours after an intravenous injection of nearly a fatal dose of eucupin none can be demonstrated in the tissues, and none has been excreted during this period, which indicates that eucupin is destroyed rather than excreted. The onset of anesthesia from eucupin is delayed because of its slow diffusibility. For this reason, and also because of the fact that the injections cause some initial burning sensation it is customary to employ eucupin in combination with procain for infiltration anesthesia. Solutions of eucupin stronger than 0.2 per cent should not be injected because local tissue reactions may occur.

Quinin and its derivatives have been used for local anesthesia in otolaryngology as early as 1911, when Ephraim[13] made submucous injections of one per cent quinin and urea in the nose. However, disagreeable hemorrhages, due to vasodilatation (in spite of using adrenalin) discouraged his further experiments. For topical anesthesia Ephraim used quinin dihydrochloride carbamate in a one per cent solution, together with epinephrin, as a spray in the posterior nasopharynx and claimed good results. We[14] used eucupin for surface anesthesia in laryngoscopy and bronchoscopy shortly after the war in one to two per cent aqueous solutions. However, a superficial irrita-

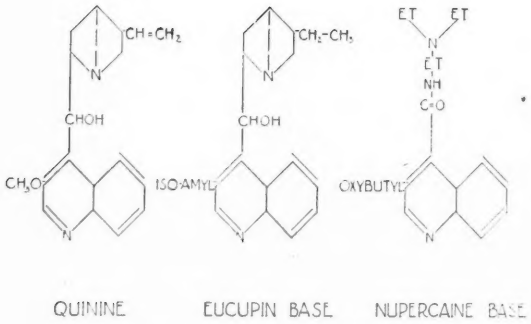

Fig. 1. Eucupin, modification of quinin molecule.

tion and slight opacity of the mucous membrane did not encourage our experiments at that time, although we were able to obtain a perfect anesthesia. Dr. Homer A. Trotter of Buffalo, New York, according to Dr. Robert H. Fowler[15] uses quinin and urea and states that a single well-placed injection of one-half of one per cent is sufficient. One cc. to each tonsil with three drops of adrenalin added to the total volume—1¼ cc.—and 15 minutes wait is required.

In 1928 another quinin derivative which has proven to be a very powerful local anesthetic was given to the medical profession. This is nupercain,[16, 17] a hybrid of quinin and procain, containing the quinoline ring of quinin and the diethylaminoethanol radical found in many of the novocain series. Nupercain (Fig. 1) is a-butyloxycinchonic acid diethylaminoethylene diamide hydrochloride. It is largely used in rhinolaryngology as a surface anesthetic.

In 1926, Francis W. Gowen of Philadelphia[18] published his paper on tonsillectomy using nerve blocking anesthesia, in which he described his method of blocking the medial and posterior palatine nerves as well as the glossopharyngeal nerve. I have used this method since 1927 in a slightly modified way in a great number of cases with very satisfactory results.

Early in 1937 we began some clinical investigations with eucupin.* Remembering my experiences of fifteen years ago, I

*Supplied through the courtesy of Rare Chemicals, Inc., Nepera Park, N. Y.

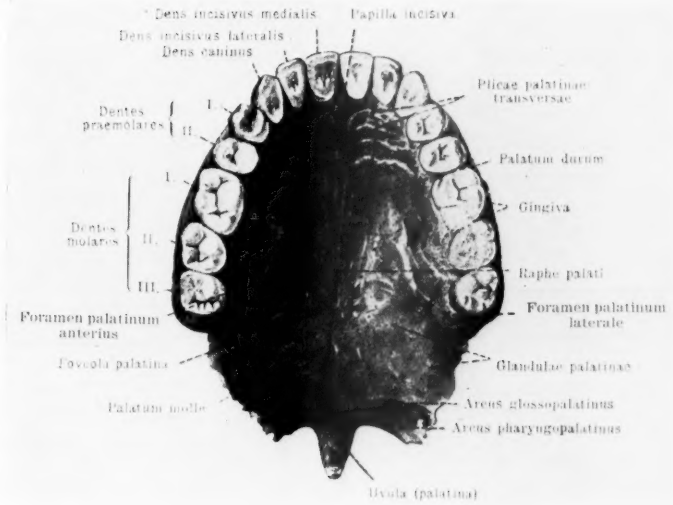

Fig. 2.

approached the problem with some misgiving, but when I learned that de Takats[8] had proved that isotonic solutions of eucupin did not produce sloughing I realized that our previous failures were due to our using eucupin in hypertonic solutions. The solution used consisted of Ringer's solution containing eucupin dihydrochloride 0.1 per cent and 0.2 per cent and procain hydrochloride 1.0 per cent, to which we added immediately before its use 15 drops of epinephrin 1/1000 to each 30 cc. of the solution. The posterior pharyngeal wall as well as the tonsillar region was swabbed with two per cent pontocain solution. We injected two ccs. of the eucupin solution at the posterolateral corner of the hard palate in order to block the medial and posterior palatine nerves as they emerge from the lateral palatine foramen (Fig. 2). This point of injection is located at the angle formed by the hard palate, the posterior end of the alveolar process, and the hamular process of the internal pterygoid plate of the sphenoid bone. Even though there are no visible landmarks this point is easily found, because the hamular process may be felt through the mucous membrane by means of an applicator armed with a cotton sponge. The five fila-

ments of the glossopharyngeal nerve which form the tonsillar plexus or circulus tonsilaris are anesthetized by the injection of five ccs. of solution posterior to the lower pole of the tonsil. To expose this latter point of injection the tongue is depressed downward medially and forward. A shallow depression then appears behind the anterior pillar and its connection with the tongue. The tonsil is now grasped with a mousetooth forcep and pulled out of its bed. With a straight needle the anterior pillar at its lower end is punctured. Advancing the tip of the needle slowly the anesthetic solution is injected into the retrotonsillar area until the level of the posterior tonsillar pillar is reached (Fig. 3). As the injection proceeds the syringe is raised and lowered so that the anesthetic solution is distributed in a fanlike manner reaching the inferior and superior filaments as well as small branches of the lingual nerve which sometimes supply the lower pole of the tonsil.

There are two advantages to this technique: First, the mucous membrane of the mouth is punctured at only two points, each of which are painted with a 2.5 per cent tincture of iodine immediately before injection; second, the needle does not pierce infected tonsillar tissue. This technique is of particular advantage in the opening of peritonsillar abscesses or in the performance of tonsillectomy in the presence of acute tonsillar infections in case this may be deemed advisable.

The danger of injuring the nerves and blood vessels in the sphenomaxillary fossa is remote if we maintain the position of the needle parallel to the lower teeth. The tip of the needle is easily controlled and the posterior pillar as well as the outer fascia of the middle pharyngeal constrictor muscle should not be pierced. By controlling our injections in this manner it is possible to avoid such fatalities as have been published by Seeger,[19] who reported a case of death occurring after an ordinary local anesthesia in which the anesthetic was injected into the carotid artery. An anomaly of the carotid artery extending toward the midline would be more easily injured by the paratonsillar injections than by the nerve-blocking technique.

We have never observed Horner's syndrome, which is associated with sinking in of the eyeball, ptosis of the upper eyelid, slight elevation of the lower lid caused by paralysis of the cervical sympathetic nerve as has been described by Meurman.[20] In Meurman's cases Horner's syndrome disappeared after one hour, the patient being as unaware of its disappearance as of its onset. Meurman, however, was

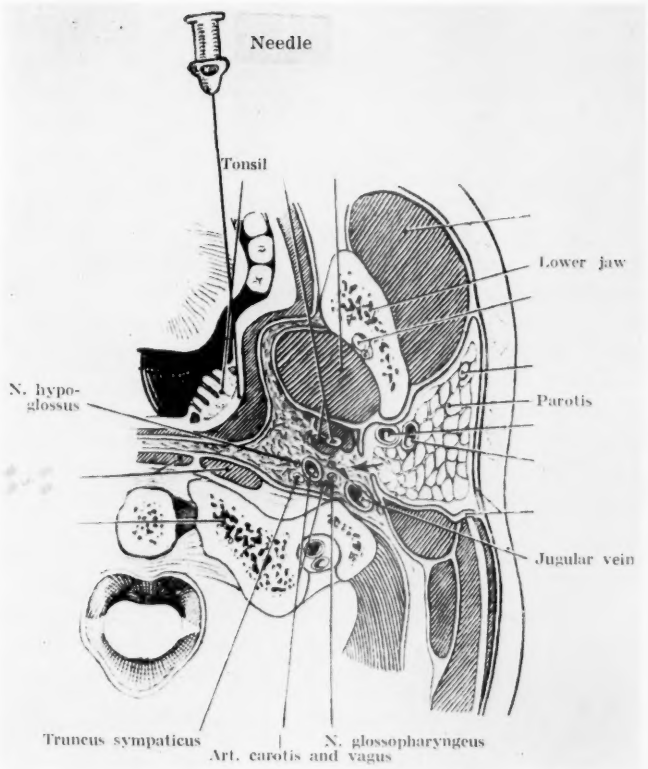

Fig. 3. Horizontal section through the level of the tonsil, showing the topographic relation of the spatium paraphayngeum to the tonsil. This section is somewhat more cranial than the plane of injection for the lower pole of the tonsil. (According to Corning's topographic Anatomy.)

later able to avoid the occurrence of Horner's syndrome by keeping his injection needle parallel to the lower teeth.

SUMMARY

Eucupin-procain solution has been used in about 50 cases at the New York Ophthalmic Hospital and College and in private practice. In a few early cases there was rather intensive bleeding which was afterward controlled by the addition of adrenalin. We now add 15 minims of a 1/1000 solution of epinephrin to each 30 ccs. of eucupin-procain solution. In the beginning of our series we injected one per cent procain with adrenalin into the right side of the throat and eucupin-procain solution into the left side, the patient being unaware that two solutions were being used. The onset of anesthesia was the same on both sides, the action of procain being neither accelerated nor inhibited by eucupin. We formed the impression that the depth of anesthesia was greater on the side in which eucupin was used. Following operation a definite difference between the two sides was observed. All the patients claimed that the eucupin side was less sore and painful on the evening following operation and in most cases this comfort lasted until the following morning. The duration of analgesia following eucupin-procain injection was from 18 to 36 hours with an average of 24 hours. In the first 20 cases eucupin 0.1 per cent was used, and in the later cases the concentration was increased to 0.2 per cent. Our impression was that analgesia was deeper and more prolonged with 0.2 per cent solution than when 0.1 per cent solution was used. In no case, after we had begun the use of 15 minims of 1/1000 of epinephrin to each 30 ccs. of solution, did we notice any greater bleeding than we have been accustomed to observe with procain solution alone. Two cases may be of interest:

REPORT OF CASES

CASE 1.—Mrs. H. B. age 30, whose coagulation and bleeding time was prolonged, and who first was treated with alleged coagulants with marked success. Tonsillectomy was performed under nerve block anesthesia with eucupin-procain solution 2.5 cc. to the posterior palatine foramen and 5 cc. at the level of the inferior pole of the tonsil. There was no excessive bleeding during operation and to our agreeable surprise the operation was rather bloodless and the patient had analgesia on both sides which lasted about 36 hours.

CASE 2.—Miss J. B., age 27, suffered from a recurrent peritonsillar abscess on the left side. Tonsillectomy was performed under nerve block anesthesia, 2.5 cc. eucupin-procain solution being injected into the lateral palatine foramen and 10 cc. below the lower tonsillar pole. Tonsillectomy was bloodless and the patient was relieved from a peritonsillar pain immediately after the operation. Analgesia lasted for about 40 hours, and the patient made an uneventful recovery.

CONCLUSIONS

1. Local anesthesia by the nerve block technique is generally superior to the paratonsillar regional injection.

2. There is the great advantage that the mucous membrane is punctured only twice on each side rather than the four or more punctures usually made with the regional technique.

3. By injecting the anesthetic solution with the needle parallel to the lower teeth the nerves and vessels of the sphenomaxillary fossa are avoided.

4. Eucupin-procain solution produces an analgesia lasting an average of 24 hours postoperatively without delay in healing or without induration of the peritonsillar tissues.

667 MADISON AVENUE.

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LXXXIV

THE ADVANTAGES OF A SPLIT SKIN GRAFT FOLLOWING TONSILLECTOMY

HAROLD I. HARRIS, M.D.

HOLLYWOOD

That "we are the product of our environment," and that "necessity is the mother of invention," have long been proverbs of the first order. They still possess their significance in fostering a great number of ideas.

Being situated in a community whose chief industry necessitates the existence of a group of professional artists, among whom a large proportion depend upon their voices as a livelihood. The members of this group, including public speakers, actors and singers of both the stage and the screen, naturally take every precaution to safeguard their voices.

It is especially the singers who are most apprehensive about their voices. They are most anxious about any type of disease affecting their throats, and rightfully so, since their entire being depends upon that special gift, the voice.

However, it is often that these people, like many others, are subject to throat affections or some systemic condition for which a tonsillectomy is a necessary part of the therapy. When so informed, there is always objection to the procedure on the grounds that it might interfere with their singing voice. The story of knowing someone who lost his singing voice as a direct result of a tonsillectomy is always related. Some patients are even more abrupt, stating that they know of their need for a tonsillectomy, but wish to know what effect it will have on their singing voice.

It was this constant query that prompted me to make a thorough study of the after-effects of tonsillectomy, and, if possible, to find some way of avoiding them.

Evidently, the subject was not one of sufficent importance to cause the writing of many reports, since a complete search of the literature was almost fruitless. The only reference found was in "The Tonsils," by Harry A. Barnes, M.D., C. V. Mosby Co., 1923.

His remarks on the "Effects of Tonsillectomy on the Singing Voice" are as follows: "Provided that no undue injury is done to the palate or to the faucial arches, the singing voice is not injured by the radical removal of the tonsils. On the contrary, a marked improvement often results. Any injury to the pillars that results in cicatricial contraction interfering with the movement of the palato-pharyngeus muscle, or any injury that leaves the palate less free than normal in its backward and forward play, has a distinctly deleterious effect upon the voice. The function of the palato-pharyngeus muscle is to tilt the thyroid cartilage upon the cricoid cartilage, thereby stretching the vocal cords. It is, therefore, an important regulator of the pitch of the voice. Cicatricial contractions between the pillars may so interfere with the movements that the quality and pitch of the voice, especially of its higher tones, may be materially modified. In discussing the question, however, we assume that the integrity of the palate and the pillars have not been affected by the operation. With this condition fulfilled, I have seen nothing but benefit to the voice result from the operation."

With the exception of the last statement, I am fully in accord with the above quotation. Even with the fulfillment of the above conditions, the fact that the normal healing that follows tonsillectomy results in a pathological structure and not in a perfect anatomical, physiological structure, is the basis for the development of this new surgical procedure.

Barnes, on page 205 of his book, in the chapter on Post Operative Deformities, states: "In uncomplicated cases, deformities of the palate and the faucial pillars should never occur. If the sinus tonsillaris is deep and the tonsillar root extensive, the surface denuded by the removal of the tonsil will include nearly the whole of the sinus, even to the edge of the faucial pillars. Under these circumstances, contraction of the cicatrix may pull the pillars so closely together that they appear to be adherent.

To this statement I must take exception. The relation of the size of the remaining pillars and the size of the tonsillar fossa, immediately following and immediately before tonsillectomy, to the subsequent size of these structures several months after the tonsillectomy, is about two to one. The elasticity of the pillars is lost. There is definite scar tissue formed on the lateral side of the tonsillar fossa, as well as that lining the denuded area of the pillars. It is therefore a direct contradiction to his previous statements, in which he states that there is no impairment of the quality and pitch of the singing voice.

It is generally conceded that the majority of people who have their tonsils removed are not particularly concerned with the effects upon their voice. It is also conceded that nearly all the tonsillectomies performed by laryngologists leave the throat in good anatomical relationship. In spite of this fact, the manner of healing has nothing to do with the method used in operating; that is out of the control of the operator.

In principle, the present method of tonsillectomy is entirely an anomalous surgical procedure. Nowhere in the body do we operate and leave a large, raw area to cicatrize, without attempting to cover the area.

I fully realize the criticism to which I am laying myself open by proposing a modification of technique to a comparatively simple operation, which would immediately change that operation to a more difficult procedure with a great many obstacles and the possibility of frequent failures.

The first criticism that might be offered is that there is so little change in the singing voice following a well-performed tonsillectomy that there is no need to subject a patient to the extra inconvenience, the possibility of failure, and the added expense. The only answer I can give is that the artist who is fearful of even the slightest change in his voice is more than willing to undergo the inconvenience associated with the taking of a graft and and sewing it into place. The possibility of failure is great. There is always infection present in the oral cavity to contend with. That is a point that must always be made clear to the patient. Even if the second part of the operation is a failure, the patient is in no worse a condition, aside from the inconvenience and added cost, than if he had undergone the conventional simple tonsillectomy. Every one that I have thus far contacted does not consider that these are sufficient reasons against having the operation done.

As is evident in the postoperative observation, the left tonsillar fossa (in which the inlay was placed) is deeper and higher, and the pillars are definite and distinct. The opposite side is shallow, and almost completely obliterated. On direct examination, the lining of the left fossa simulates a mucous membrane covering, while the right side is definitely scar tissue. Feeling of the glosso-pharyngeus and the palato-glossus muscles shows a definite difference of the two sides; the left is more soft and pliable than the right. The subject does not happen to be a singer, but if the change in voice is dependent upon the change in anatomy, no matter how slight, there must be some alteration in the pitch, if not in the quality of the voice.

This operation has a further advantage in that before one can put a graft on the tonsillar bed, all oozing must be completely stopped, and the dissection must be done with extreme care.

My first operation was done with novocain, infiltration anesthesia, but I should suggest that those doing the operation for the first time do it under general intratracheal anesthesia.

There was a two-fold reason for doing the operation on only one side. First, I have been nearly six months trying to find someone who would permit me to operate thus on their throat, and secondly, because I felt that having the tissues of one individual as a control would render a more accurate basis for comparison of tissue reaction.

The procedure following the usual removal of the tonsil is: one must be certain that there is a complete hemostasis, then dental molds of the respective tonsillar fossa are made. Three grooves are made on the medial aspect and an area at the mid-point on the medial aspect is crushed which is subsequently used to grasp the inlay for introduction into the fossa after the graft is placed upon the inlay. Through this center elevation, a hole is made to pierce the area crushed, which can be threaded with a silk suture to prevent the swallowing of the stent, in case some unforeseen situation should arise.

A split skin graft is taken from the inner side of the thigh, one side or both, sufficiently large to cover the two molds made. The molds are covered, with the raw area of the skin graft placed toward the tonsillar fossa. Three right angle mattress sutures are made, one near the top of the fossa, one near the base, and another at approximately the middle. The stent is grasped with the tonsillar forceps by the groove made for that purpose, and placed in the fossa, to be held there by an assistant while the sutures are tied with No. 2 plain catgut. The edges of the skin graft are approximated to the pillars, and several interrupted catgut sutures are placed, attaching the two. These are left in place for about ten days, when they are ready to fall out.

The patient is fed liquids only, through a glass tube. When the stent comes out, it will be noticed that the graft has adhered to the walls of the sinus tonsillaris. The fossa is the same size as immediately after the tonsillectomy, and several months later, the contraction is practically nil in comparison to the contraction normally observed after the conventional method of tonsil removal.

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The Scientific Papers of the American Bronchoscopic Society

LXXXV

BRONCHO-ESOPHAGOLOGY: POSTULATED SEMASIOLOGIC OBSERVATIONS*

CHEVALIER JACKSON, M.D.

PHILADELPHIA

It is with diffidence that I beg to call the attention of this Society to a matter that seems to me worthy of its consideration.

When this association was organized, 21 years ago, the bronchoscope and esophagoscope had opened an untrodden field for clinical observation and research, had rendered possible the visual examination and direct study, in the living, of the anatomy, physiology, pathology, and treatment of the tracheobronchial tree, the esophagus and stomach. At that time we were busily engaged in looking into the air and food passages. The name, "Association of American Peroral Endoscopists," was above criticism from the semasiologic viewpoint. Our society was a group of "lookers in through the mouth." From the practical viewpoint, however, the name was a clumsy one to use; so much so, in fact, that by unanimous action four years later the name was changed to The American Bronchoscopic Society. This was a great euphonic betterment without semantic detriment. We were still a society interested in looking in through the mouth?

But do we not, today, do more than simply look in?

If we are interested only in viewing, the adjective, bronchoscopic is correct; if we are interested in the etiology, pathology, symptomatology, diagnosis, prophylaxis and treatment of diseases of the tracheobronchial tree the word is a semasiologic solecism. The Greek suffix *skopion* (from Greek *skopein,* modern Latin, *scopium*) means view-

*Abstract of an address delivered at the meeting of the American Bronchoscopic Society, Atlantic City, April 30, 1938.

ing; it does not include the scientific knowledge acquired by viewing. It seems to me that in the 21 years that have elapsed since this society was founded we have accumulated a mass of scientific knowledge that merits the suffix logos. With the usual combining vowel the word would be "bronchology." This word would cover the branch of medical science that deals with the tracheobronchial tree, but as a name for our society it would cover only one phase of our work and would leave unremedied another feature of our present name, Bronchoscopic, that is questionable; it does not do justice to the great amount of work that has been done on the esophagus and its diseases. At the time this society was founded we did little more than look into the esophagus. Since that day Dr. Mosher and others have placed diseases of the esophagus on a scientific basis. Though the esophagoscope has contributed in an invaluable way to this work there is much of it that is not esophagoscopic. It seems to me that to hide this mass of knowledge under the adjective Bronchoscopic in the name of our society is not only an injustice to this branch of medical science, but additionally it is a semasiologic solecism. The addition of the adjective "esophagoscopic" would not adequately correct the injustice, nor would it correct the solecism, because it means only the instrument or the viewing; it does not mean the knowledge acquired. It seems to me that the knowledge of the esophagus and its diseases, as developed, accumulated and recorded by Dr. Mosher and other workers in and outside of the membership of this society merits the suffix *logos*. With the combining vowel we have the word esophagology as covering a department of study and a branch of medical science. This would naturally lead to the compound word broncho-esophagology as combining the two main fields of work. If this be granted, a logical name for this association would be the American Broncho-Esophagologic Society. As a new name it may sound a little awkward, but is it not as smooth and manageable as the name American Gastro-Enterologic Association?

A number of questions naturally arise:

Shall we be accused of trying to magnify our importance?

Is the word bronchologic in the dictionaries?

Does the accumulated knowledge in the respective branches justify the appellations bronchology and esophagology?

What have we to consider in the way of precedent and analogy?

Shall we be accused of trying to magnify our importance?

We certainly shall. Precisely that same accusation has been brought forward against each and every specialty in turn. From

ophthalmology in the last third of the last century down through all the -ologies to roentgenology in recent years, all specialties have had to face this criticism. When the first laryngologic society was formed in 1873 criticism was bitter. Dr. J. Solis Cohen was expelled from the Academy of Natural Sciences for daring to call himself a laryngologist. Roentgenologists were first called radiographers, and were by some rated as a mere subclass of photographers. By their work they soon demonstrated their right to the term roentgenologist and established the specialties of roentgenology and radiology.

However, criticism is wholesome. It is better not to meet it with controversy, but simply to plod along, piling up the data of our research and clinical work. The tracheobronchial tree and the esophagus are both vital viscera; the advantages of special departments of study devoted to them cannot be denied.

Is the word bronchologic in the dictionaries?

No, it is not. It might be added that the word bronchoscopic was not in any dictionary at the time this society adopted it for a name. It is not in some of them yet. The fact of the matter is that lexicographers follow, they do not lead; and they lag long laps behind the literature. No dictionary is ever up to date no matter how often new editions appear. I had taken nearly a hundred foreign bodies out of the bronchi with the bronchoscope before the name of the instrument first appeared in any dictionary. A dictionary is simply a compilation of the words in the literature of the language concerned. Lexicographers do not create words. In general, the people create them, often, but not always, in form of slang at first; in literature authors create them; in medical science, physicians create them. In all of these classes the new words may be created out of old words or parts of words already in the same language or in a foreign tongue; or they may be created *de novo*. In the sciences the new words are usually based upon Latin or Greek roots, ordinarily with due regard to established usage as to declensions, plurals, prefixes, suffixes, etcetera. A scientific body may take up the subject of nomenclature and make order out of chaos by authorizing certain words, creating others, establishing a system by which words newly created from time to time can be formed to fit in the established system. For example, the science of chemistry was in such a tangle that meanings were not clear, spelling was varied, inconsistencies were absurd, errors were not infrequent; to avoid these time and space were being wasted by the cumbersome undisciplined form of words that had been haphazardly absorbed into chemical literature in course of time. The American Chemical Society brought order out of chaos

and established a code by which future new words could be consistently coined and fitted into chemical literature and verbal usage. The lexicographers have not yet caught up to the American Chemical Society, but eventually they will.

Does the accumulated knowledge justify the term bronchology, or the term esophagology?

The more I see of the inside of that bronchial tree the more I feel it deserves a department of medicine devoted to its study. I feel the same way on looking into the esophagus. But this is from only a personal viewpoint; justification of the word requires a broader consideration. The best evidences that can be offered are (a) our program for today; (b) our proceedings covering a fifth of a century of progress, as enumerated by our President, Dr. Berry, this morning; (c) the researches and clinical data recorded in the medical literature of the world. If all this material evidence could be collectively presented on library tables it would be conclusive as to the existence of a science of broncho-esophagology as distinguished from the art of broncho-esophagoscopy. Additional evidence is the fact that Mr. Norman Patterson, after attending one of our meetings a few years ago, expressed the opinion that our society should be called bronchologic rather than bronchoscopic; a compliment rather than a criticism, I consider it.

Precedent and Analogy.— It is pertinent and convincing to consider the nomenclature of other societies an important part of whose work is endoscopic. The most striking thing is that we are the only -scopic association. There is no telescopic society; if there is any science based on an instrument it is astronomy. Nor is there any national society designated as spectroscopic, microscopic, fluoroscopic, stethoscopic, enteroscopic, otoscopic, rhinoscopic, pharyngoscopic, laryngoscopic, opthalmoscopic, proctoscopic or cystoscopic. All of the societies using the instruments that gave rise to these adjectives have adopted the suffiix, -ology, because it represents the knowledge whereas the ending -scopic refers to the procedure; knowledge relates to the science, the procedure is an art. There was once an American Microscopic Society. As a member I happen to know that before it disintegrated the eminent scientists composing its membership decided that there was no science of microscopy.

In order to get an abstract view of the inappropriateness of the name bronchoscopic as applied to our society let us imagine suggesting to the American Ophthalmological Society that it change its name to the American Ophthalmoscopic Society.

In suggesting for consideration the changing of the name of this society it should be emphasized that it is not a matter of terminologic hairsplitting, nor is it a matter of semantics; but rather of (a) the establishment of a new department of study; (b) doing justice to our work; (c) conforming to the act of all other special medical societies in adopting a name indicating the field of work, or department of study, rather than the method or the instrument of examination.

There is another reason, and I think it is an important one. The name of our society entirely ignores one of the important fields of our work, namely, the esophagus.

Finally, if this society should decide that Bronchoscopic is a *nomen conservandum*, I should venture to predict that at some future time the membership as then constituted will reverse the decision. Moreover, I should still believe that there are departments of study worthy of the names bronchology and esophagology.

LXXXVI

LARYNGOCELE*

W. Likely Simpson, M.D.

Memphis

A laryngocele is an air sac connected with the larynx. Sometimes this condition is spoken of as a pneumatocele and again as an aerocele.

Jackson,[1] in discussing the laryngocele, speaks of it as an analogue of a similar structure normally found in gorillas, apes and chimpanzees, but rarely there are aeroceles of the larynx which do not depend upon preformed structures. The usual laryngocele has its origin from a dilated ventricle of Morgagni, which was described by Virchow[2] in 1867, and is an enlargement and extension of the ventricle, probably taking place by way of the sacculus, and is lined with mucous membrane which is covered with ciliated epithelium.

This sac may be either intralaryngeal or extralaryngeal. There is also the mixed type in which the sac is both intra- and extralaryngeal and is joined by a small isthmus. The extralaryngeal portion is often quite large. Usually if the sac has become extralaryngeal it has protruded through the thyrohyoid membrane. It may have protruded through the cricothyroid membrane, or very rarely, following an injury or a pathological process making a pathway, it may be in any area in the larynx.

Garel[3] divides laryngoceles of the larynx into superior and inferior—the superior being those at the level of the thyrohyoid membrane, and the inferior those at the level of the cricohyoid membrane. He makes a further division into sub-glottic aeroceles and tracheoceles. Jackson[4] speaks of air sacs sometimes developing below a chronic laryngeal stenosis.

Laryngoceles may develop in normal larynges without any preexisting pathological condition, following such etiological factors as singing, horn-blowing, glass blowing, weight lifting, vomiting, childbirth, coughing, whooping cough and politzerization. Such conditions as syphillis and tuberculosis, tumors and other diseases affecting the

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*Presented before the meeting of the American Bronchoscopic Society, Atlantic City, April 30, 1938.

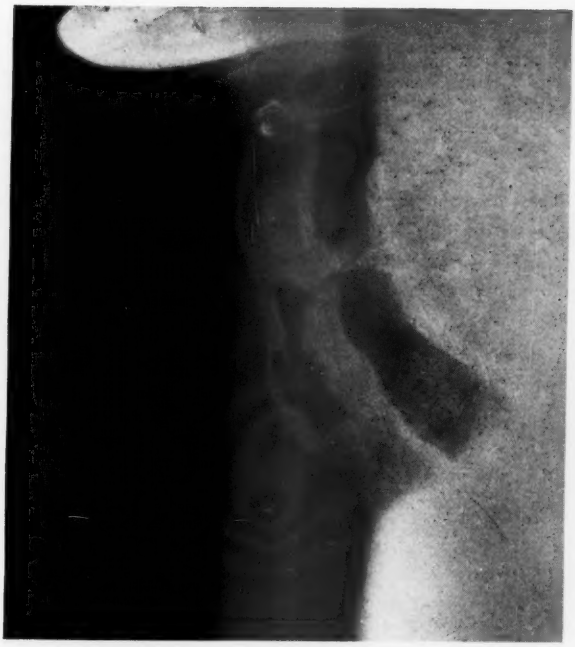

Fig. 1. Roentgenogram demonstrating air in the laryngocele.

larynx may weaken its walls in such a manner that an aerocele may arise, especially if the above mentioned etiological factors are also present.

The air sacs formed in diseased larynges may not have a definite wall lined with ciliated epithelium as does the anomylous type of laryngocele. In recent traumatic cases, such as those mentioned by Broca[5] and Scheven[6] and in my own case which I am reporting, there was not a definite sac lined with ciliated epithelium.

Garel[3] says that even though laryngoceles which arise in pathological larynges and from trauma do not have definite walls, a diffuse emphysema does not arise.

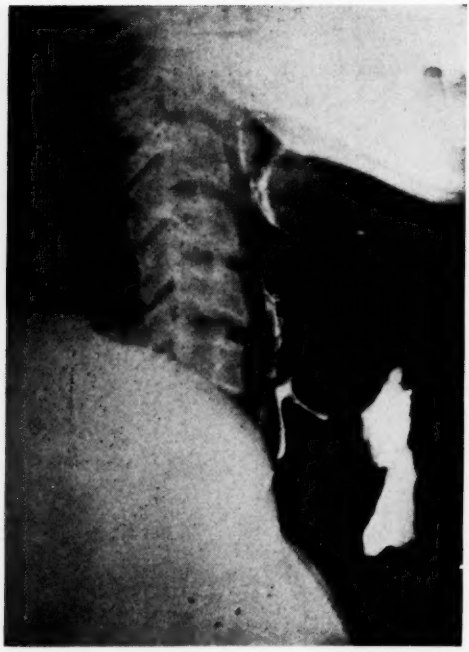

Fig. 2. Roentgenogram with lipiodol in the laryngocele.

Anyone who wishes to make a study of laryngoceles of the larynx would do well to read the article by Irwin Moore,[7] who discusses the subject very thoroughly and has made a study of the 85 cases which were reported from the time of Moxon's case in 1868 until the time his paper was written in 1920.

Since 1920 there have been quite a number of cases reported. Jackson[8] and Iglauer[9] of this society have reported cases.

My own case is that of Mr. V. J. S., 32 years of age, who came with a history of injury to the larynx region and hospitalization for one week two years previously. There was a small mass at the midline

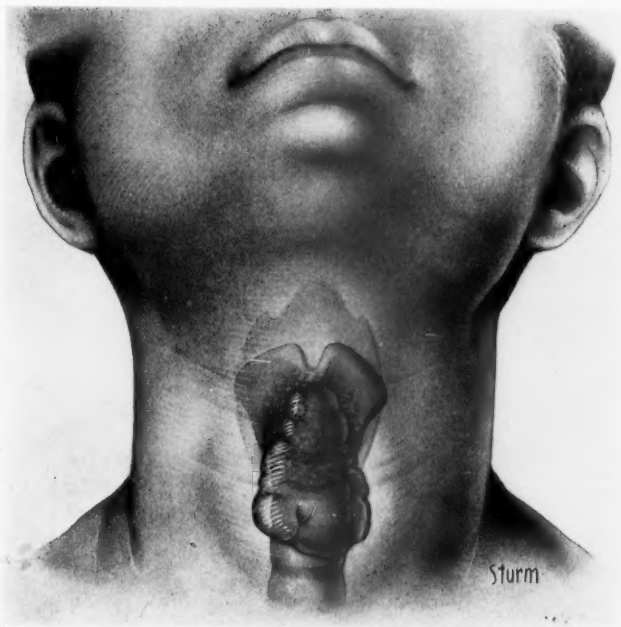

Fig. 3. Schematic drawing of anterior view of the neck showing the laryngocele before the operation.

of the larynx region up until about 10 days before my examination when this region became larger and slightly red.

An examination showed a red swollen area one inch in height and an inch and a half in diameter on the anterior surface of the neck at the junction of the cricoid and trachea. In changing the position of the head and neck and upon compression of this area there was a peculiar noise as if air was escaping through a small opening. This phenomenon was demonstrated several times.

Under local anesthesia a midline incision was made over the laryngeal and tracheal region. A mass reaching from about the

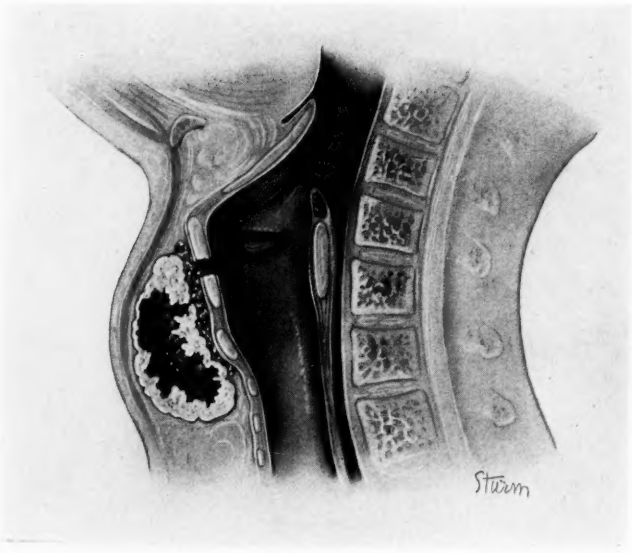

Fig. 4. Schematic drawing of lateral view of the neck showing the laryngocele before the operation.

second ring of the trachea upward well onto the thyroid cartilage was uncovered. It was not well outlined and was opened during dissection. It contained a small amount of straw-colored fluid and air and had a thick wall and a central cavity of about four drams in size. From this cyst cavity there was an opening into a second cavity which extended over both wings of the thyroid well down onto the cricoid. The wall of this second cavity seemed to be made up of granulation tissue. There was an opening leading from this second cavity through the middle third of the right wing of the thyroid through which a probe could be inserted upward into the hypopharynx. The cystic mass most anteriorly situated was entirely removed by dissection. The second cavity which was surrounded by a granulation tissue wall was thoroughly curretted. The opening in the larynx was closed by means of a muscle flap. The entire wound was closed in the usual manner and an uneventful recovery took place.

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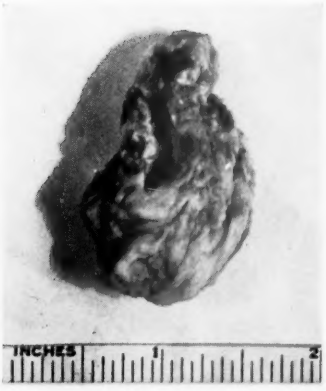

Fig. 5. A photograph of the laryngocele after removal. The black area shows the opening from the laryngocele into the granulation space.

Fig. 6. A photograph of the opened laryngocele after removal.

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LXXXVII

BRONCHOSPIROMETRY*

GEORGE R. BRIGHTON, M.D.

AND

ALVAN L. BARACH, M.D.

NEW YORK

The double bronchoscope is not a new instrument, but it is a new instrument in this country. It was developed by Dr. Paul Frenckner in Sweden in the Nose and Throat Clinic of the Sabbatsberg Hospital in Stockholm in conjunction with the late Professor Jacobeus[1] in 1932. This instrument was devised for the purpose of separating the respiratory air of each lung for simultaneous analysis. The term which has been used to describe this procedure by Jacobeus, Frenckner and Bjorkman[2] is Bronchospirometry, which is an easier way of expressing the more cumbersome term Bronchoscopic Spirometry. The description of the instrument as set forth by Frenckner[3] is as follows: "The double bronchoscope consists of two tubes for respiratory air, two tubes for illumination and one tube for inflating the rubber caps. This last has an extremely fine lumen, allowing the passage of only a small stream of air. The tubes for lighting purposes are similar in thickness to those in Jackson's standard bronchoscope. The air tubes must fill the above-mentioned requirements in order that sufficient quantities of air may pass per unit of time and with the same ease as physiologically occurs. Theoretical calculations show that a tube of the required length should have an inner diameter of 6 mm. for men and 5.5 mm. for women to be satisfactory. By making these tubes slightly oval and suitably connecting them with the three others, the outer diameter of the larger bronchoscope would be nearly 13 mm. and of the smaller, at the most 12 mm.

"One of the respiratory air tubes, with its lamp carrier, runs along the instrument from the proximal to the distal end, and is intended for the left main bronchus. The most distal centimeters of the instrument are transformed into the distal or lesser obturator.

*From the Nose and Throat Department and the Department of Medicine, College of Physicians and Surgeons, Columbia University and the Presbyterian Hospital.

Presented before the meeting of the American Bronchoscopic Society, Atlantic City, April 30, 1938.

The other respiratory air tube and its lamp carrier ends 7 to 8 cm. proximal to the distal end, and is intended to be placed right above the carina to exchange air from the right lung. The whole of the instrument just above the distal end of the shorter respiratory tube is cylindrical, and it forms the proximal or thicker obturator, which is intended to form a closure against the walls of the trachea a centimeter or two above the carina. At the proximal end of the double bronchoscope there are two outlets for respiratory air, one on either side, two illumination attachments for the lamp carriers and one device for inflating the rubber caps. The proximal ends of the respiratory air tubes are fitted with conical window plugs, which permit control of the contents of the tubes and whatever may be at the distal end of the bronchoscope during the entire investigation.

"Bronchoscopic instruments necessary at a bronchospirometry, apart from the double bronchoscope, are mainly an equipment according to Jackson. The ordinary Jackson laryngoscope for adults is, however, some millimeters too small in its inner diameter, therefore a somewhat larger size is required.

"This is then the equipment needed by the endoscopist in order to obtain air from the right and left lung separately for delivery to the analyst.

"For his analyses a double spirometer which can take respiratory air from the left and right lung simultaneously, and register volume and oxygen consumption is necessary."

The patients are prepared in the usual way for bronchoscopic examination. They are admitted into the hospital in the morning and after a period of rest in bed for an hour or two, they are given morphine and one of the barbiturates approximately an hour before bronchoscopic examination. Morphine and scopalamine are added when the patient is sent to the operating room. The larynx is cocainized in the usual way by topical application to the superior laryngeal nerve in the pyriform fossæ. A special laryngoscope is used to expose the larynx and the double-barrel bronchoscope is passed through the laryngoscope into the larynx and the distal obturator is passed into the left main bronchus. When the distal obturator is seen to pass the carina into the left main bronchus the window plugs are inserted into the bronchoscope and both obturators are inflated. In our technique the obturators were inflated by using a constant oxygen pressure which was determined before the bronchoscope was passed so that when the bronchoscope is in place a pressure valve is opened from a constant oxygen source and the obturators are dilated.

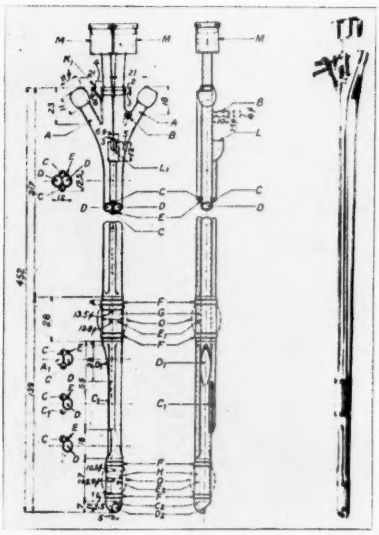


Fig. 1. The double bronchoscope and its construction. *A*, Outlets for the respiratory air. *B*, Olive for the attachment of the rubber hand bulb for inflating rubber cap. *C*, Tubes for illumination. C_1, Location of the proximal illumination. C_2, Location of the distal illumination. *D*, Tubes for the respiratory air. D_1 and D_2, Their distal orifices. *E*, Tube for inflation of the rubber caps. E_1 and E_2, Its distal orifices. *F*, Metal rings for the attachment of the rubber caps. *G*, The proximal obturator. *H*, The distal obturator. K_1, Device for fixing the light carrier proximally. L_1, Device for fixing the light carrier proximally. *M*, Window plugs. *O*, Rubber caps.—From: Proceedings of Royal Society of Medicine, Section of Laryngology.

The first picture is taken from the works of Frenckner,[1] which shows the double bronchoscope or as we have termed it, the double-barrel bronchoscope.

In the following drawing a schematic representation of the double-barrel bronchoscope showing the method by which the obturators are blown up to fulfill their respective positions. It is important that one be sure that the proper pressure is maintained so that none of the inspired air escapes during expiration around the instrument. In our experimental procedures instead of using the bulb sphygmomanometer method we use a constant oxygen pressure

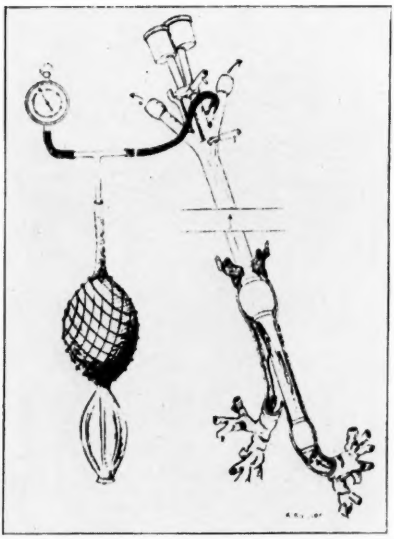


Fig. 2. The double bronchoscope in position.—From: Proceedings of the Royal Society of Medicine, Section of Laryngology.

which has seemed to be more satisfactory. We have also reproduced a photograph of the double spirometer which was used in Stockholm. Our machine is very similar to this.

In the first graph the pulmonary ventilation of a relatively normal individual is represented. This patient was admitted to the hospital for persistent coughing. He was 60 years old and in fairly robust health. The bronchoscope was passed and we were unable to find any evidence in the tracheobronchial tree of any pathology to explain the cough except perhaps a slight congestion on either side of the carina. Bronchospirometric readings were made. This curve goes from right to left. The vertical lines on the graph represent time and the distance between each vertical line equals one minute. The upstroke of the curve is inspiration and the downstroke expiration. The depth of the curve represents the tidal volume for each inspiration. The average tidal volume is determined by a measurement of the depth of each upswing and downswing. The parallel lines on the graph represent units of oxygen. The distance between each parallel line represents two units of oxygen in cubic centimeters

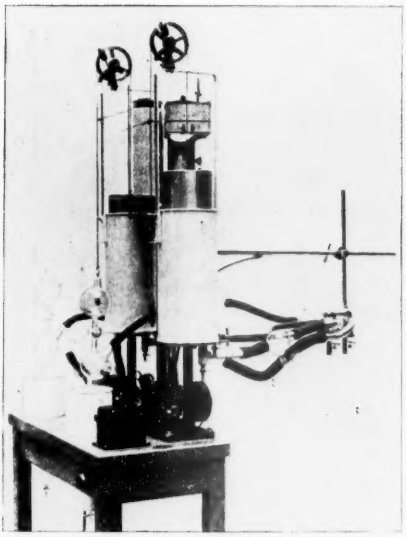
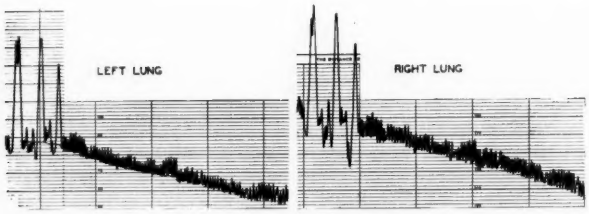


Fig. 3. The double spirometer.—From: Proceedings of the Royal Society of Medicine, Section of Laryngology.

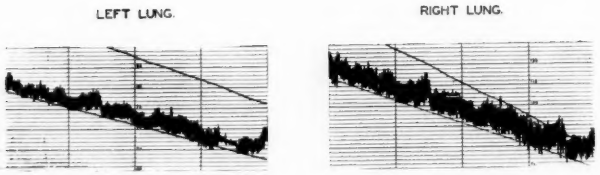
and each unit is valued at 20.73 cc. of oxygen. Therefore, one can see that the minute volume of oxygen in cubic centimeters is the tidal air times the respiratory rate. The large swings on the graph represent deep inspirations and expirations or the vital capacity of each lung. This graph then represents by its inclination the oxygen consumption of the individual in both the right and left lungs for a period of over four minutes. By taking the total oxygen consumption of both lungs we can express in percentages of absorption the right and left lung separately. The normal figures which were made in the Stockholm clinic by bronchospirometric examinations on medical students show that the right lung usually consumes more oxygen than the left and the ratio, expressed in percentage of total oxygen consumption is given usually as 52% of the total consumed in the right lung and 48% in the left lung.

In this patient, despite the persistence of a troublesome cough, graphic records (Fig. 4) of the ventilation of the right and left lung reveal no abnormality in lung function in respect to the volume of air breathed per minute, the oxygen consumption, and the relation



148 CC.	TIDAL AIR	181 CC.
21	RESP. RATE	21
3110 CC.	MINUTE VOLUME	3800 CC.
146 CC.	OXYGEN CONSUMPTION	180 CC.

Fig. 4.



136 CC.	TIDAL AIR	205 CC.
41	RESP. RATE	41
5580 CC.	MINUTE VOLUME	8470 CC.
151 CC.	OXYGEN CONSUMPTION	212 CC.

Fig. 5.

286 CC.	TIDAL AIR	475 CC.
24.5	RESPIRATORY RATE	24.5
7,000 CC.	MINUTE VOLUME	11,650 CC.
179 CC.	OXYGEN CONSUMPTION	292 CC.

Fig. 6.

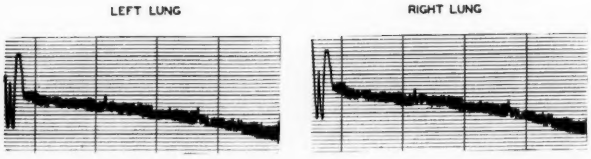
of these factors between the two lungs. It will be seen from the graph that the vital capacity of each lung may be calculated separately. In studies which are contemplated on a fast moving drum, the velocity of air movement in each lung may be more readily calculated.

We have included a patient with bronchial asthma. The patient is 58 years old and has had bronchial asthma for a very long time. He has been bronchoscoped many times because he gets considerable relief for a fair length of time after each bronchoscopy. Bronchoscopic examination shows simple edema of the mucous membrane throughout the tracheobronchial tree.

A bronchospirometric curve (Fig. 5) was made of this patient during one of the bronchoscopic examinations and treatments for his asthma. There are several interesting observations to be made here. The first one is the rapidity of the respiratory rate. The patient's respiratory rate is 41 respirations per minute. His tidal air volume, as you will see, is 205 cc. in the right side and 136 cc. in the left. However, when we express his oxygen consumption in terms of percentage rather than cubic centimeters, we find that his right lung is absorbing 59% as compared with 41% in the left lung.

The total pulmonary ventilation in this case was 14 liters per minute, which is slightly more than twice the normal volume. The right lung itself had a minute volume of 8470 cc. It is of considerable interest to observe that the tidal air on the right side was 205 cc. as compared to 136 cc. on the left side, suggesting a much greater narrowing of the smaller bronchi on the left as compared with the right side. It may be pointed out in addition that the pulmonary ventilation is more increased than would be indicated for this patient's oxygen consumption. This type of hyper-ventilation in asthma as a manifestation of obstructive respiration is not uncommon.

Bronchospirometric observation shows that the oxygen absorption in the left lung is considerably less than in the right lung. The left lung obsorbed only 38% of available oxygen with relatively normal respiratory rate on both sides. Paralleling the relative decrease in oxygen consumption, there was a much smaller tidal air and total pulmonary ventilation in the left lung. As you will notice, in this patient we were able to get only a small curve because of the considerable amount of secretion which welled up in the left lung. However, if you will look at the curve for one full minute you will see that there is apparently no obstruction to the normal inflow and outflow of air in that minute. The respiratory excursions seem fairly constant.



103 CC.	TIDAL AIR	105 CC.
25	RESP. RATE	25
2580 CC.	MINUTE VOLUME	2620 CC.
72 CC	OXYGEN CONSUMPTION	74 CC

Fig. 7.

179 CC.	TIDAL AIR	160 CC.
21	RESP. RATE	21
3760 CC.	MINUTE VOLUME	3370 CC.
188 CC.	OXYGEN CONSUMPTION	82 CC.

Fig. 8.

The next patient had pulmonary tuberculosis and was referred for bronchoscopic examination because of repeated hemoptosis. Because her sputum was negative it was felt she might have some other lesion. When she was examined it was found that she had moderate stenosis of the right upper lobe bronchus. The x-ray examination shows some evidence of old tuberculosis on the left side.

As you will see from the bronchospirometric tracing (Fig 7), each lung consumed an almost equal amount of oxygen. The oxygen absorption in the right side is 49% and in the left 48% with a respiratory rate of 25 respirations per minute, and a total pulmonary ventilation of 5200 cc. per minute, an essentially normal (or low normal) figure.

The bronchospirometric readings on a patient who had a lobectomy on the right side reveals that there is available some lung tissue on the right side, but apparently because of previous pulmonary tuberculosis on that side there is very little oxygen absorption. As may be observed on the graph (Fig. 8), the inclination of the oxygen absorption curve in the right lung is very low, whereas in the left lung it is quite steep. The right lung is absorbing less than 39% of the oxygen available.

CONCLUSION

The technique of employing the double bronchoscope for the study of the pulmonary ventilation of each lung is described. The procedure is capable of providing accurate determination of the function of each lung in respect to oxygen consumption, the volume of ventilation and the velocity of air movement. Previous results reported in Sweden make it seem likely that this test has a clinical usefulness, such as the demonstration of an adequate lung function in cases of pulmonary tuberculosis in which radical collapse of the opposite lung may be contemplated. Furthermore, the field of pulmonary physiology is extended by this method which has made possible the separation of the ventilatory behavior of each lung.

580 PARK AVENUE.
893 PARK AVENUE.

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LXXXVIII

THE ROLE OF INFLAMMATORY BRONCHIAL STENOSIS IN THE ETIOLOGY OF BRONCHIECTASIS*

PAUL H. HOLINGER, M.D.

CHICAGO

Bronchiectasis is a disease characterized by one of several types of bronchial dilatation accompanied, usually, by varying degrees of pulmonary suppuration. In the late stages of bronchiectasis the bronchial walls show extensive destructive changes. These are preceded by inflammatory manifestations which appear to be an important phase of the early stage of the disease and give rise to the process which leads to the development of bronchiectasis. The specific reaction in which we are interested at this time is the inflammatory bronchial stenosis which produces complete or partial bronchial obstruction leading eventually to atelectasis and then to bronchiectasis.

The object of this paper is twofold: first, to correlate this sequence of bronchial inflammation, bronchial obstruction, atelectasis and, eventually, bronchiectasis as it has been observed in a series of children; second, to compare a previously reported series of bronchiectatic children who were observed from the onset of the pulmonary disease and followed to fully developed bronchiectasis with similar cases observed early and treated bronchoscopically, in which the process was aborted; thus, to re-emphasize the effect of breaking this sequence by prompt recognition and clearing of the original lesion.

The relationship between atelectasis and bronchiectasis has been known for many years. Andrus,[1] after carefully studying all the more commonly accepted theories in regard to the development of bronchiectasis, concludes that "atelectasis is probably a causative agent of high frequency in bronchiectasis, and constitutes the most rational explanation as yet available of the otherwise obscure or idiopathic

*From the Bronchoscopic Clinics of The Children's Memorial Hospital, St. Luke's Hospital, and the Research and Educational Hospital of the University of Illinois College of Medicine, Chicago.

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causes of this disease." Lee Lander and Davidson[2] further emphasize the importance of this relationship, stating that atelectasis is the most important factor in the production of bronchiectasis. The review of the literature in regard to this point is especially complete in these two monographs.

Atelectasis as a definite clinical entity has, peculiarly, been one of the least understood conditions of the chest. The first accurate description differentiating atelectasis from pneumonia was given by Louis[3] over 100 years ago, in 1829. Fundamental work on the etiology of this condition is surprisingly old. Mendelssohn[4] in 1845 suggested bronchial obstruction as the principal etiologic factor involved, and produced atelectasis by placing gum arabic into the bronchi of animals. Gairdner,[5] in 1850, also believed that bronchial obstruction was the chief factor. This work was forgotten to some extent, however, when in 1890 William Pasteur[6] of England suggested that paralysis of the diaphragm and chest wall was the predominant etiologic factor. To support this, he presented 15 cases of diaphragmatic paralysis followed by atelectasis in patients with diphtheria. Pasteur's theory remained foremost for several decades. It is true that atelectasis is seen in cases of acrodynia and pseudo-hypertrophic muscular dystrophy, as well as in postdiphtheritic diaphragmatic paralysis. While paralysis is still recognized as one of the etiologic factors in the production of atelectasis in certain cases, it is now recognized that the atelectasis is produced in these cases because stagnant secretions obstruct the bronchi. More recent experimental and clinical evidence supports the work of the early observers who demonstrated that by far the most common cause of atelectasis is bronchial obstruction, most dramatically shown in cases of foreign bodies[7] and postoperative massive collapse of the lung.[8] Obstruction of a bronchus is now recognized as the chief cause of acquired atelectasis.[9]

In 1934 Anspach[10] reported a series of 50 cases taken from a group of 100 in which a triangular shadow had been observed at the base of one or both lungs for periods up to 12 years. Clinically, the areas presenting such a roentgenogram were found to be dull to percussion, and auscultation revealed bronchial breathing, bronchophony, or silence.

Such shadows and findings frequently led to a diagnosis of pneumonia, unresolved pneumonia, mediastinal or interlobar empyema, or atelectasis. Some of the shadows cleared within a short time, and in those cases the course of the "pneumonia" was limited,

and there was a clinical return to normal which paralleled the roentgen clearing. Some shadows disappeared, recurred, and finally cleared entirely, while still others remained dense more or less permanently. Nine cases came to autopsy, and in each the triangular shadow was found to be a collapsed lower lobe. The most important group in regard to the relation between atelectasis and bronchiectasis consisted of patients in whom the triangular shadow remained present several months to a year or more. Patients in this group were found to have developed bronchiectasis. In studying the records of 50 of these bronchiectatic children, such histories were frequent and of extreme importance. It was apparent that many of the children had been examined both physically and roentgenologically during a prebronchiectatic stage and followed until a definite bronchiectasis was established. Thus, Anspach correlated the triangular shadow with atelectasis and demonstrated by serial x-rays and postmortem findings that bronchiectasis developed in the atelectatic areas if they remained atelectatic.

During the past two and one-half years, a majority of these cases have been studied again in the Bronchoscopic Clinic of The Children's Memorial Hospital, and additional information has been gained regarding the intrabronchial pathology.

The histories of a number of these patients are surprisingly similar. In a few of Anspach's cases the collapse was caused by a foreign body, glandular compression of the bronchus, granulation tissue, or tumor. These are not included in the present discussion. In the great majority of his cases, however, the clinical course was that of a mild pulmonary infection, thought at the time to be pneumonia. While the acute phase was generally short and aberrant, the symptoms failed to clear up, and the physical findings persisted. The patient developed a cough and continued with it in spite of appearing to be in fairly good health. Associated with this persistence of physical findings and cough, x-ray revealed the presence of the triangular shadow. In the far advanced cases, this type of history was repeated many times; with each attack the collapse was of longer duration, and finally became permanent. By that time, the destruction of the bronchial walls had taken place; the triangular shadow had become more dense, and bronchography revealed the many sacculations which demonstrated that bronchiectasis had developed. This was, of course, an end stage, and it was found with surprising frequency in children as well as adults.

In an attempt to determine the beginning of the process, we have arranged the cases from Anspach's series which we have examined,

with additional similar cases, according to the severity and duration of the disease. The earliest cases which could be included were found to be those in which an upper respiratory infection with associated pulmonary findings occurred for the first time. In these patients, the pulmonary pathology resembled pneumonia, but analysis of the clinical findings and course, together with the roentgen picture, demonstrated that the process was one of atelectasis. There was a persistence of dullness, bronchial breathing, bronchophony, and rales on one side, with a shift of the heart and mediastinal structures toward the involved side and an elevation of the involved diaphragm. The roentgen picture confirmed these findings, demonstrating a triangular shadow at the base of one lung, usually less dense than that seen in the far advanced cases (Fig. 1).

In most of the cases in which the collapse occurred for the first time, it was found to clear spontaneously, but recovery was speeded by rigidly enforcing postural drainage, with or without the use of expectorants. "Cough mixtures" were definitely contraindicated. The material coughed up during postural drainage was always thick, tenacious, and purulent. Such cases were considered definitely "pre-bronchiectatic" because the tenacious secretions which the child was trying to expel had obstructed a bronchus and produced a temporary atelectasis. Thus, these apparently simple cases of aborted pneumonia, or "unresolved pneumonia," are the earliest cases of atelectasis which Anspach has shown eventually develop a far advanced bronchiectasis if the atelectasis is allowed to remain. "The determining factor (as to which cases go on to bronchiectasis) in the great majority of instances is mechanical obstruction."[11]

The entire future course of the disease is determined at this stage. As Clerf[12] says, "Retention of pus will ultimately lead to bronchiectasis. For this reason, if for no other, early recognition of bronchial obstruction is important." The difficulty lies in recognizing the importance of the seemingly insignificant physical findings. As Miller[13] states, if "efforts at prevention would be directed particularly to the care of the apparently slight pulmonary affections during the early years of life" the appalling frequency of bronchiectasis would be reduced. The difficulty in making a diagnosis at this stage of the disease is due to the fact that the child appears to have entirely recovered from his acute "pneumonia." He is up and about, usually playing, but has a persistent cough and a low-grade temperature, which remain in spite of the administration of cough mixtures, which are given only too frequently in these cases. The cough should be

Fig. 1. Radiographs showing early triangular shadow at the right base which persisted two months following an "atypical pneumonia." The child had a cough and a low-grade temperature, with physical findings of dullness and bronchial breathing at the right base.

stimulated rather than retarded. Jackson,[14] Thorpe,[15] Clerf,[16] Brennemann,[17] and others have repeatedly stressed this fact.

Cases in which symptoms persist in spite of adequate medical management show little change in the roentgenogram from week to week. Such cases, as Ferguson[18] points out, require bronchoscopic aspiration in order to re-establish aeration and prevent destruction of the bronchial walls within the atelectatic area. The bronchoscopic picture in such patients is of extreme interest and of utmost importance, because the striking pathology found in the tracheobronchial tree during this stage can be treated to bring about a definite change in the course of the disease. The trachea and opposite main bronchus are usually normal, although they may show a slight degree of inflammation. On the involved side, however, the bronchus is found to be considerably inflamed and to contain a moderate amount of thick, viscid pus. Aspiration of this reveals an edematous, stenotic bronchial orifice which leads directly to the area shown by physical examination and x-ray to be atelectatic. This orifice is usually intensely red and almost occluded by the inflammatory swelling. Frequently thick, white, tenacious pus can be seen exuding or oozing from the bronchus on cough, with no air bubbles in the pus, indicating further that air neither enters nor leaves this portion of the lung. Obviously, only a slight degree of mucosal inflammation, together with this heavy pus, is necessary to block the tiny bronchial divisions of a child.

Following the initial bronchoscopy, in which the cycle of inflammatory obstruction, infection, and increased obstruction was broken, a further large percentage of these early cases cleared and remained well (Fig. 2). As a matter of fact, prompt relief of symptoms and disappearance of the lesion followed bronchoscopic aspiration in the cases which were bronchoscoped early. We can still consider these cases definitely "pre-bronchiectatic." Every effort must be made to clear them as quickly as possible because we noted repeatedly, on the other hand, that in those cases in which the lung had remained atelectatic over a longer period of time, it required an actual as well as proportionately longer period of time to reaerate the lung (Fig. 3). In cases in which the atelectasis remained present for a period of months, repeated bronchoscopic aspiration and bronchial dilatation were necessary to re-establish normal function (Fig. 4).

From our series of cases we noted that, when a portion of the lung had been collapsed several years, frequent bronchoscopic drainage generally failed to restore normal function to the atelectatic

Fig. 2. Radiographs of same patient as shown in Fig. 1, following bronchoscopy. Dilatation of the inflammatory and bronchial stenosis and aspiration of the obstructing purulent secretion, which produced the atelectasis, resulted in prompt reaeration of the involved portion of the right lower lobe.

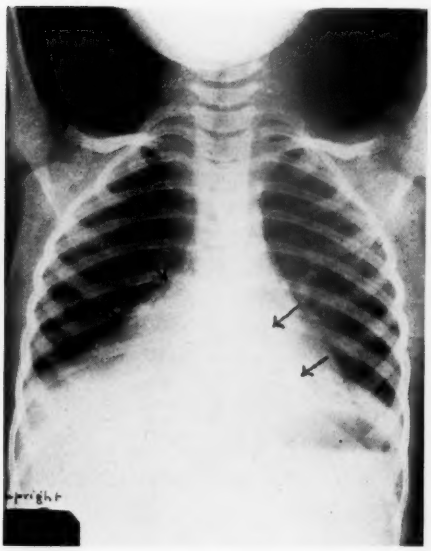
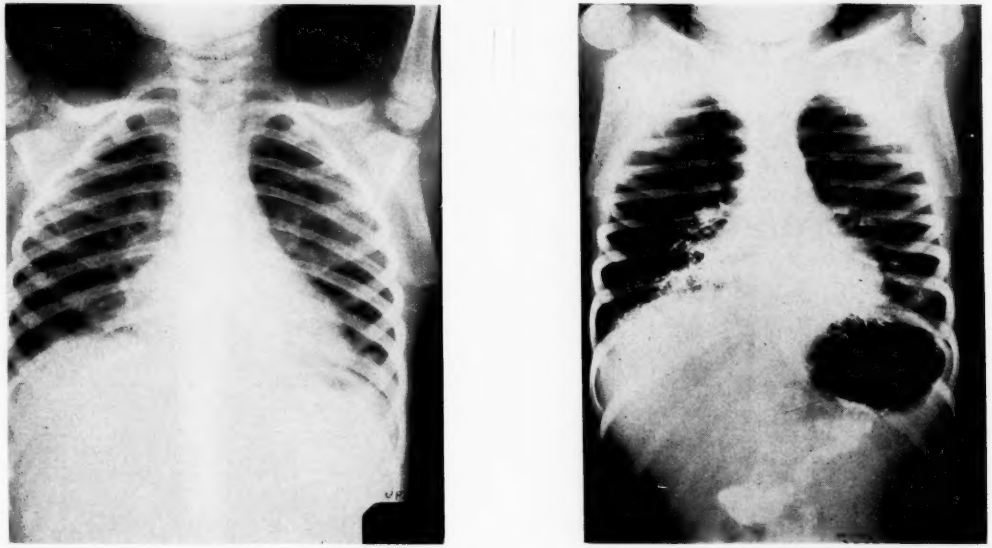


Fig. 3. Radiograph showing bilateral triangular shadows in a four-year-old girl. Atelectasis has been present for at least a year.

lung. Such therapy did, however, markedly reduce the amount of sputum and change its foul character. Similarly, it reduced the amount of pneumonitis of the adjacent pulmonary tissue. Such therapy aided in improving the child's general condition sufficiently to lessen appreciably the surgical risk, since lobectomy was definitely indicated. (Fig. 5.)

Comparative studies of the bronchographics of this series of patients demonstrate another extremely important point.[19] Lee Lander and Davidson[2] demonstrated both experimentally and clinically that there is a bronchial dilatation which occurs as soon as the lung becomes atelectatic. In our cases in which drainage was established early by either medical management or one or two bronchoscopic aspirations, and in which the observed duration of the inflammatory stenosis was relatively short, subsequent bronchography revealed a normal tracheobronchial tree (Fig. 4-B). With an increase in duration of the atelectasis, however, in spite of satisfactory reaeration of



(A) (B)

Fig. 4. Radiographs of same patient shown in Fig. 3 after repeated aspiration of obstructing bronchial pus and dilatation of inflammatory bronchial stenoses. There has been a re-aeration of the atelectatic lower lobes (A), and the bronchography shows the size and distribution of the bronchi to be normal (B).

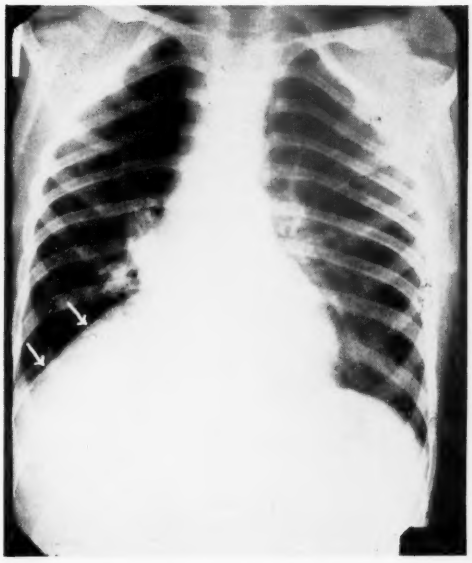


Fig. 5. Radiograph of a nine-year-old boy with an atelectatic triangular shadow at the base of the right lung, present many years. History of repeated attacks of "pneumonia."

the lung, bronchography showed beginning dilatation of the bronchi in the involved area. Thus, the residual pathology (bronchiectasis) was found to be roughly proportional to the duration of the collapse. (Fig. 6.)

CONCLUSIONS

We have stressed the diagnosis and treatment of prebronchiectasis, i.e., before the pulmonary disease has reached the stage in which there is permanent destruction and dilatation of the bronchial walls. This stage has been recognized by correlating various clinical and roentgen findings of children with advanced bronchiectasis who have been studied during the past ten years. They are arranged in sequence in relation to the course of the disease from the advanced stage back to the earliest mainfestations of pulmonary pathology. The particular type of bronchiectasis studied was that associated with atelectasis.

Fig. 6. Bronchography, postero-anterior and right lateral views, of the previous case (Fig. 5), demonstrating the far advanced bronchiectasis present in the atelectatic triangle. Repeated bronchoscopic aspiration reduced the pneumonitis around the affected area and aided in a palliative manner, but did not alter the bronchial dilatation.

"Prebronchiectasis" has been characterized in our cases by an area of dullness, bronchial breathing, rales, a chronic cough, and a low-grade temperature, which may have followed an upper respiratory infection or a so-called pneumonia. Persistence of such findings is of utmost significance since x-ray reveals an atelectatic or partially atelectatic triangular shadow in such cases. (Fig. 1) The bronchoscopic picture is that of an acutely inflamed bronchial orifice, stenotic, from which pus oozes without bubbling. By shrinking the mucous membrane around that orifice with cocain, by dilating it with forceps, and then by passing an aspirator directly into the orifice, pus can be released and normal function eventually restored. (Fig. 2) Subsequent lipiodol reveals a normal bronchial tree. This type of case, as is shown in Anspach's series, eventually develops typical bronchiectasis if untreated.

In cases of atelectasis of longer duration (Fig. 3) an attempt should be made bronchoscopically to open the airway and permit better drainage of pus. After repeated bronchoscopy, reaeration may be accomplished. (Fig. 4) In other cases showing triangular atelectatic areas which had been present more than a year, there were definite bronchiectatic cavities which marked lung destruction and bronchial dilatation (Figs. 5 and 6). Aspiration aided these patients in reducing the inflammatory reaction and in a palliative manner, but could not return normal function. Medical as well as fairly regular bronchoscopic treatment failed to change their progressive course. At this stage, if the process is unilateral, we must resort to surgery.

SUMMARY

1. Various stages in the development of one type of bronchiectasis from the prebronchiectatic atelectasis to the clinically well-known far advanced stage are presented in sequence.

2. Inflammatory bronchial stenosis was found to be responsible for the production of the original pulmonary lesion, atelectasis.

3. "Atelectasis precedes and plays a prominent and most constant role in the development of a common form of bronchiectasis of the lower lobes."[10]

4. Persistent pulmonary findings simulating pneumonia are frequently due to atelectasis. If the lung does not clear spontaneously, early and frequent bronchoscopic drainage of the involved bronchi will prevent permanent dilatation and destruction of the bronchial walls, which follow if the atelectasis is left untreated.

707 FULLERTON AVENUE.

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LXXXIX

RUPTURE OF THE ESOPHAGUS WITH SPONTANEOUS RECOVERY OF THE PATIENT*

SAMUEL IGLAUER, M.D.

CINCINNATI, OHIO

The possibility of spontaneous recovery after rupture of the esophagus is so remote that I feel justified in reporting the following case which presents some very unusual features:

History: The patient, a colored girl, aged 6 years, was admitted to the hospital March 21, 1936. The day before she had swallowed a lye solution, and on admission showed erosions of the mouth and pharynx. Dysphagia was extreme at first, but gradually subsided under expectant treatment.

At the time of discharge four months later (July 16, 1936) she could swallow all sorts of food, and x-rays taken after barium ingestion showed no definite point of obstruction, but a rather uniform narrowing of the esophagus. The patient was instructed to return to the clinic for further observation.

She did well until April 6, 1937, when she was brought back to the hospital, unable to swallow anything but water. X-ray examination on the following day showed a diffuse stenosis of the esophagus beginning at the level of the aortic arch and extending down to about an inch or two above the diaphragm, with some dilatation above the strictured area. The barium in the dilated area appeared mottled as though mixed with food.

Following hospitalization she began to swallow spontaneously without any special treatment, and was discharged at the end of a week with orders to return in three weeks, or sooner if necessary.

August 8, 1937, the patient was brought to the hospital for the third time. She was unable to swallow anything.

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*From the Department of Otolaryngology, College of Medicine, University of Cincinnati.

Presented before the meeting of the American Bronchoscopic Society, Atlantic City, April 30, 1938.

Examination: X-ray visualization of the esophagus with lipiodol revealed an almost complete obstruction at the level of about the fifth dorsal vertebra. From this point downward the lumen was almost threadlike in character, while above the obstruction there was an irregular dilatation. Esophagoscopy showed that the upper end of the esophagus was occluded by a practically impassable cicatrical stricture having a one-millimeter opening.

Operation: The patient was transferred to the Surgical Department, and a successful gastrostomy was performed under ether anesthesia on August 11, 1937.

Treatment and Course: Following this operation the patient was fed through a gastrostomy tube. One month later traces of methylene blue taken by mouth appeared in the stomach, and after another month this patient finally succeeded in swallowing a string.

On October 10, 1937, retrograde dilatation was undertaken by one of the residents. A No. 12 F. soft rubber catheter was pulled through without difficulty. A No. 14 F. was then tried, but the string broke under traction. Within the next 24 hours there was a rapid rise in the pulse to a maximum of 160, with a temperature of 103°+, and a respiratory rate of 40. October 12, 1937, physical and x-ray examinations, aside from revealing a deviation of the trachea to the right, were essentially negative. The large gastric feeding tube was delineated in the stomach.

Four days later there were signs either of consolidation or effusion in the lower left lung area. The x-ray films at this time and later showed the distal end of the gastrostomy tube *definitely above the diaphragm lying in the posterior mediastinum behind the heart.* (Figs. 1 and 2.) At this period the child began expectorating foul-smelling mucopurulent material.

I was asked to see the patient on October 19, 1937. It was my opinion that a mediastinitis was present and that the tube had made its way through a rupture of the lower end of the esophagus and diaphragm (?) into the posterior mediastinum. I advised against immediate removal of the tube since it appeared to me to be acting as a drain from the mediastinum into the stomach. I also requested consultation with the Surgical Department to consider the advisability of a posterior mediastinotomy.

The following tests were then made: (1) Dr. Moore found that air blew out through the gastrostomy tube with each expiration,

Fig. 1. The distal end of the gastrostomy tube is seen in the posterior mediastinum behind the heart. The tube has been filled with lipiodol, some of which has entered the bronchial system.

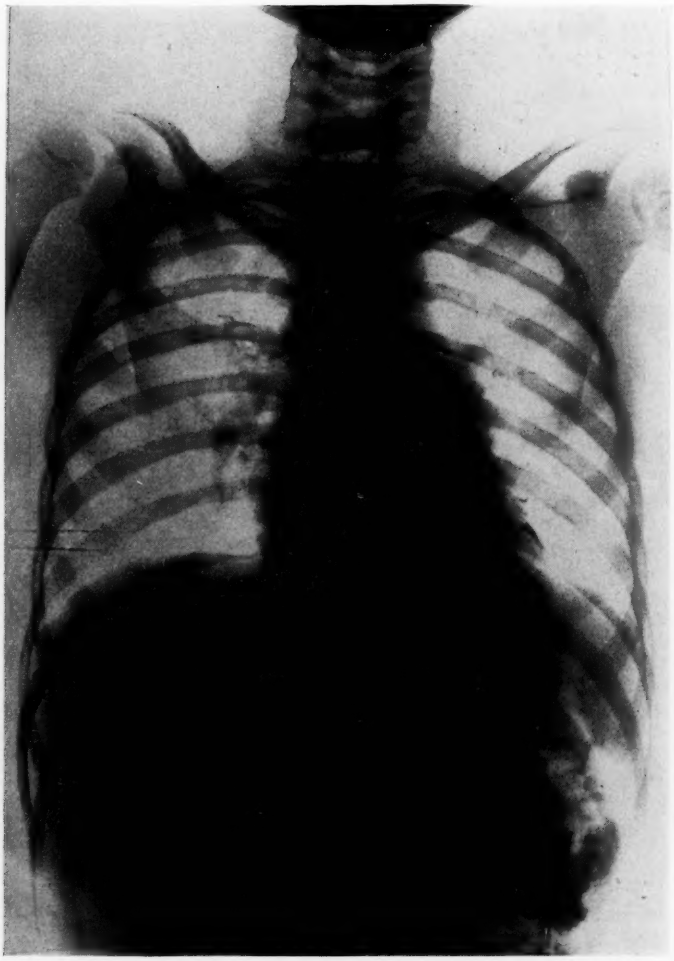

Fig. 2. A. P. view confirming Fig. 1. The presence of lipiodol in the left bronchus gives conclusive evidence of the presence of a fistulous tract extending from the stomach through the mediastinum and into the bronchial system.

indicating a connection with the bronchial system; (2) the stomach contents were aspirated, and contained purulent foul-smelling material similar to the sputum previously expectorated; (3) the gastrostomy tube was filled with lipiodol, and the oil was seen to enter the bronchial system (fluoroscope), giving conclusive evidence of a fistulous tract extending from the stomach through the esophagus into the posterior mediastinum and into the left bronchus. (Figs. 1 and 2.)

The surgical consultants, Drs. Reid and Carter, had not considered mediastinotomy urgently necessary, and advised the withdrawal of the gastrostomy tube and expectant treatment. Accordingly, the tube was removed on the afternoon of October 20, and a Levine tube was inserted through the gastrostomy with hope that it might enter the duodenum and thus put the stomach at rest.

During the following week, the fever and pulse gradually subsided to normal, with gradual cessation of expectoration and coughing.

The gastric tube was removed on November 2, 1937, and oral feeding was resumed. The patient was discharged from the hospital January 16, 1938. At this time she was receiving retrograde dilatation with Tucker bougies and was swallowing coarse food without difficulty. She was instructed to return at weekly intervals for further treatment.

DISCUSSION

In reviewing this case it seems certain that the lower end of the esophagus, and possibly the diaphragm, were torn during the second attempt at retrograde dilatation of the esophagus. This accident was followed almost immediately by a marked general reaction on the part of the patient. The first roentgenograms after the accident showed the gastric feeding tube in the stomach, but later pictures showed the distal end of the tube in the posterior mediastinum (Figs. 1 and 2). The tube was very long and thick, and through a sort of spring action had found its way through the fistula into the posterior mediastinum. The only other possible explanation was that the tip of the tube had exerted pressure over a period of several weeks, and had caused an ulceration of the stomach wall at its junction with the esophagus.

Following the rupture, a mediastinitis and a pleuritis must have ensued, with a fistula extending from the stomach through the esophagus and mediastinum and terminating in the left bronchus. This was proven by the injection of lipiodol through the tube and

by the appearance of the oil in the bronchial system. It should also be noted that the same sort of pus was recovered both from the sputum and from the stomach.

The gastric tube must have provided a drainage tract from the mediastinum into the stomach, and probably was instrumental in promoting the ultimate recovery of the patient.

707 RACE STREET.

XC

NEW NAIL EXTRACTING FORCEPS*

C. L. Jackson, M.D.

Philadelphia

This instrument that have to present is what I call a "bronchoscopic nail-puller." Mr. Pilling, Sr., objects to that term. I was just talking with him a moment ago, and he stated that he thought that a rather undignified name for the instrument. He prefers to call it a "nail-extracting forceps," but rather than discuss any further the naming of the instrument we shall proceed to speak of its form and the need that prompted its devising.

Dr. Jackson, Sr., has taught us all to study the mechanical problem presented in a difficult foreign body case by obtaining a duplicate of the foreign body and then trying the various methods

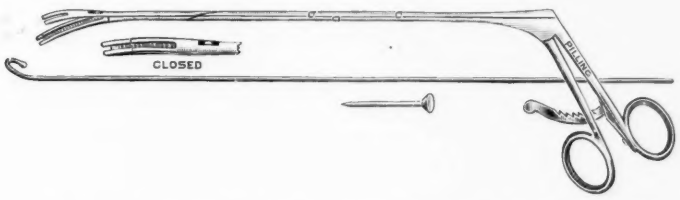

Fig. 1. Nail extracting forceps, designed for extracting nail perforating median wall of right main bronchus. A small hook was used as an auxiliary instrument, to dislodge the extreme tip. (See Fig. 3.)

that would seem to accomplish its removal without doing damage or increasing damage already done to the patient. This instrument is an example of one devised for a particular problem in a particular patient. Unlike the apparatus just presented by Dr. Holinger, it probably won't have any wide application. The instrument itself is shown in Fig. 1, but in order to understand its use it will be necessary to review briefly the case in which it was used.

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*Presented before the meeting of the American Bronchoscopic Society, Atlantic City, April 30, 1938.

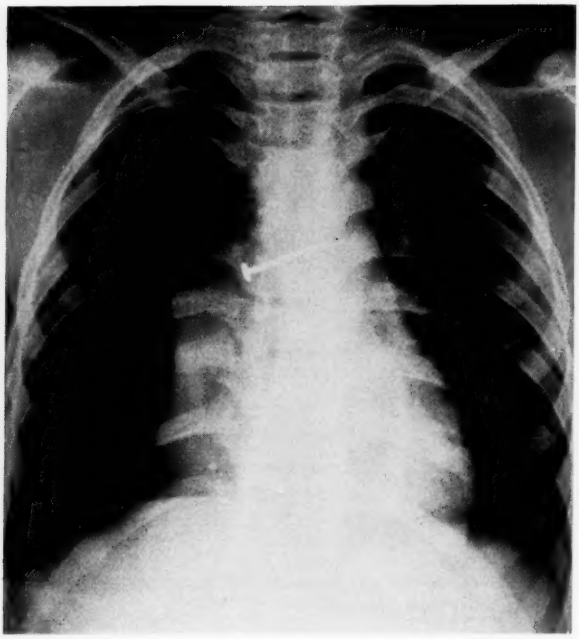


Fig. 2. Film showing nail perforating median wall of right main bronchus and causing complete right pneumothorax.

The boy came in with a pneumothorax of the right side and with the point of the nail apparently quite well out into the mediastinum, perforating the medial wall of the right bronchus (Fig. 2). The head of the nail was in the right upper lobe orifice. We allowed the reaction to subside, the lung to re-expand, and the temperature to come to normal before doing anything. Then we made an additional study to show the relation to the esophagus. It seemed the point was very close to the esophagus, but rotating the patient in another plane, we found that the nail didn't actually perforate the esophagus.

The problem was to extract this nail without causing a recurrence of the pneumothorax and without producing any lighting-up of the mediastinitis.

So considering the various ways that this might be done, it seemed that the best procedure would be to attempt to extract the

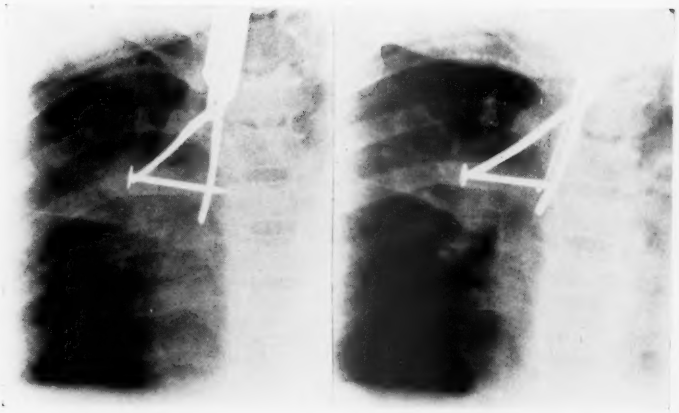


Fig. 3. Views taken on biplane fluoroscope showing, at left, the first attempt at extraction from the median wall of the right main bronchus and *(b)* second and successful attempt made the same day, after having hinged blade of forceps lengthened as well as slot in fixed blade. Note manner in which small hook was used to dislodge extreme point and bring nail into line.

nail out into the upper lobe bronchus. I used an open cadaver to test the various methods of accomplishing this result and finally devised this nail-extracting forceps. At the first attempt, under fluoroscopic guidance, we were able to extract the nail partially, but there was still too much point projecting (Fig. 3). So we sent the forceps immediately back to Mr. Pilling with the request that he make the hinged arm a little longer, and that he cut the slot which is in the fixed blade a little bit further up into the blade. The same evening, another attempt was made with this modified forceps, and we found that we were able to extract the point completely out of the bronchus. Then we introduced a small hook to free up the extreme tip of the nail and bring it into line (Fig. 3). Removal was quickly accomplished with an ordinary forceps.

The boy had one day's elevation of temperature and then it came to normal. Roentgen study about a week later showed complete subsidence of the reaction.

3701 North Broad Street.

Clinical Notes

XCI

TRAUMATIC DEAFNESS FROM THE EXPLOSION OF A FIRECRACKER—A CASE REPORT*

C. C. Bunch, Ph.D.

St. Louis

The effects of intense acoustic stimulation on the auditory mechanism are not fully understood. Fosbroke[1] in 1831 stated of blacksmiths' deafness, "It has been imputed to a paralytic state of the nerve occasioned by the noise of forging by certain modern writers, and by old writers, to a permanent overtension of the drum membrane." Stevens, Davis and Lurie[2] in 1935 reported after one of their experimental animals had inadvertently been exposed to a very loud sound, "Miscroscopic examination showed that the exposure had been sufficient to shake the organ of Corti loose from the basilar membrane." Fosbroke noted that the deafness in blacksmiths was progressive and became noticeable at the age of 40 to 50 years. Stevens, Davis and Lurie did not note the condition of the hearing of their experimental animal, but it seems probable that if deafness were produced it would have been permanent.

On the other hand, Bauer[3] in describing the deafness in aviators, says, "The constant noise of the high-powered motor causes diminution of hearing. The pilot who has no protective device will invariably be markedly deaf. This deafness gradually wears off after a few hours. Constant flying without protection results in permanent impairment of hearing." Rankin[4] stated that the deafness in aviators is, "Always of temporary nature." Scott [5] said that it lasted "From one to several hours." White,[6] who examined a woman 19 days after she had received a severe acoustic shock while using the telephone found, "No deafness, no nystagmus," and concluded, "Judging by the reports of such cases, the damage would only be temporary." Shambaugh and Knudson[7] in a report of a similar case

*From the Oscar Johnson Institute, Department of Otolaryngology, Washington University Medical School.

stated, "The deafness had been nearly complete after the shock, but hearing gradually returned." Pierce[8] reported a case in which the hearing had returned to normal after 14 days.

These divergent opinions justify the presentation of the following case report:

Case 1.—Mr. L. C., age 22. On July 5 while the patient was walking along the street, some children threw a firecracker at him. The cracker exploded close to the right side of the patient's head. (Exact distance not known.) He immediately noted very marked deafness in the right ear with some pain and tinnitus. The tinnitus was not great and seemed to fluctuate in loudness.

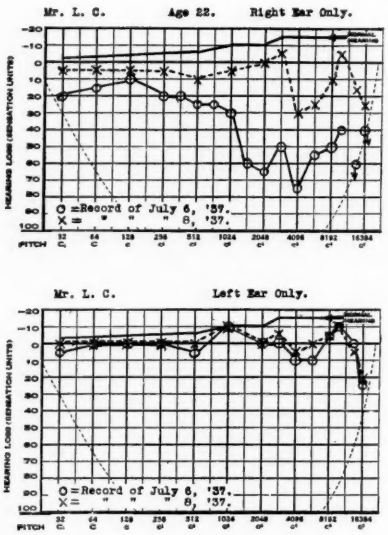
He consulted Dr. Louis J. Birsner within a very short time. Dr. Birsner found that the right drum membrane was somewhat reddened, but there was no bleeding or perforation. The patient was referred to the writer for hearing tests on the following day.

The audiometric tests (Western Electric 1A.), illustrated in the accompanying figure, show that the hearing in the left ear was essentially normal with the exception of a slight loss for tones between 2048 and 8192 d. v. The right ear showed a loss of from 10 to 20 db. for tones below 512 d. v. Tones of higher pitch showed a much greater loss, the maximum loss being near 4096 d. v. In order to determine the permanency of the hearing loss, the patient returned for a second test two days later.

The records for each ear are illustrated in separate charts. It will be noted that the curves for the left ear are almost identical. Those for the right show marked recovery. In fact, on July 8 the patient felt that the hearing in this ear was normal. However, the second test shows a loss of from 5 to 15 db. for tones below 2048 d. v. At 4096 d. v. a dip of approximately 45 db. is still present. This dip almost disappears for 8192 d. v. Whether further recovery took place is unknown. The patient failed to return for further examination as requested.

COMMENT

The positive Rinne, somewhat decreased bone-conduction, Weber test referred to the unaffected left ear and the much greater loss for high tones than for low give the classical picture of deafness of the primary nerve type. However, since the recovery was prompt and almost complete, except for the dip at 4096 d. v., it is certain



Audiograms in a case of unilateral deafness caused by the explosion of a firecracker near the right ear.

that no extensive damage could have been done to the end organ or nerve.

Concerning these dips, Fowler[9] in 1929 stated, "It would appear that mid-high frequencies were particularly sensitive to various insults, especially acoustic trauma and toxic neuritis, because they regularly fail to escape in these conditions. . . . It would appear that there is an element of toxic neuritis or trauma diagnosable by the presence of marked deafened areas (dips) and not otherwise ascertainable." Bunch[10] in 1937 wrote, "if a loss of auditory acuity has resulted from the effects of excessive stimulation of industrial noises, it is first evidenced by an abrupt dip in the hearing range as determined by the audiometer, usually near c^5 (4096 d. v.). Acoustic trauma should be suspected when these gaps appear. They are commonly found in the records of the hearing of sportsmen and hunters who often have no loss in acuity for the spoken voice and are unaware of any hearing defect whatever."

SUMMARY

This report presents the audiometric tests of a patient who was deafened by the noises of an exploding firecracker. Almost complete recovery had occurred by the third day following the accident. The case is presented as evidence which tends, in part at least, to indicate the relationship between deafness and the excessive stimulation by loud sounds.

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XCII

MYXO-FIBRO SARCOMA OF THE EXTERNAL EAR

FRANK HAND, M.D.

AND

GERALD BROWN O'CONNOR, M.D.

SAN FRANCISCO

In reviewing the literature for the last 10 years the authors were amazed at the paucity of reports on myxo-fibro sarcoma of the external ear. To date only two cases have been uncovered, one reported by J. Treer,[3] 1928, and one by V. Speziale,[2] 1932. We wish to report this case not only for the rarity of the condition and the location of the sarcoma, but for its very interesting and instructive clinical course and the operative method employed in combating the malignancy.

This individual was seen as a clinic patient by many medical men over an eight-year period and a short resume of her early history gives an interesting background.

April 5, 1927, the patient, age 5, was admitted to a local hospital with a diagnosis of right mastoiditis. No mention was made in the physical examination of a tumor of the right external ear.

April 5, 1927, right mastoidectomy performed.

April 30, 1927, right mastoid incision enlarged to provide drainage.

June 29, 1927, plastic closure of postoperative mastoidectomy.

November 30, 1929, readmitted to hospital with tumor on the peak of right auricle.

Preoperative Diagnosis: Cyst of right ear.

Postoperative Diagnosis: Fibroma of right ear.

Histological Examination: Fibroma of skin of right ear.

Treatment: Radium treatment given.

November 23, 1931, readmitted to hospital for recurrence of tumor of the right ear.

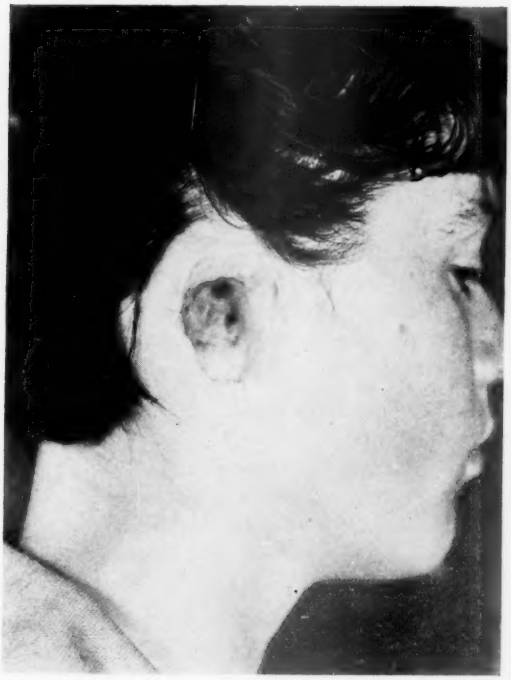

Fig. 1

History: Swelling of the upper part of the right ear; patient has had as long as she can remember; not painful; does not ulcerate.

Examination: Tumor mass, right upper helix 2 x 1 x 0.5 cm. Skin normal, no ulceration. Body of tumor mass feels like cartilage.

Preoperative Diagnosis: Chondro-sarcoma, right ear.

Postoperative Diagnosis: Chondro-sarcoma, right ear.

Operation: The helix and anti-helix above the cymba were excised almost completely. The cartilage was separated in the remaining portion from the skin, edges undercut and sufficient cartilage removed to give adequate skin flaps. A plastic was done to bring up the lower wall to join the point of amputation so as

to preserve the concha. The helix, anti-helix and the sulcus were preserved by this plastic.

Microscopic Examination: Specimen consists of upper half of right ear with several smaller pieces of blood-stained cartilage. The ear specimen measures 2 cm. in height. The base measures 3 cm. in breadth and the apex measures 2 cm. The base measures 5 mm. in depth and the apex measures 14 mm. On the outer aspect of the specimen there is a tumor which lies underneath the skin without ulceration. It encroaches slightly upon the anterior aspect and extends about 6 mm. above the edge of the ear. On the posterior aspect it extends down about 1.5 cm. On section, the tumor is a firm whitish growth showing trabeculi scattered throughout it. It extends along the cartilage and in some places appears to be attached to the cartilage. These appear to be normal except for blood staining.

Microscopic examination of the sections show the skin wheresoever found to be normal. The tumor has approached to the skin, but is not through it. The tumor consists mainly of fibroblasts and in this there are numerous aggregations of cells which seem to be forming an intra cellular cyst. The fibroblasts show occasional mitoses as do the other type of cells which approaches cartilage. In several places the tumor appears to be springing from the edge of the cartilage.

Diagnosis: Fibro-chondro-sarcoma of very slow growth.

On May 25, 1933, the patient was readmitted to hospital for recurrence of tumor of right ear.

Operation: Resection of right ear except lobe. Skin graft taken from right leg and applied under dental impression wax to denuded area of operation.

Microscopic Examination: Specimen consists of remains of ear. Four previous operations have been done. Specimen appears to be the entire remains of the auricle of the ear. This tumor occupies the whole area of that part of the ear. It appears to have no connection with the ear cartilage. On section the tumor is firm, white and fibrous. Accompanying this are a few small bits of what appear to be cartilage embedded in a blood clot.

Microscopic examination of sections show the skin to be normal. The cartilage is definitely hyperplastic. Occasional mitoses are seen. The tumor now appears to be a mixture of chondromatous and fibrous elements. Some elements resembling myxoma are seen.

Diagnosis: Myxochondro-sarcoma—grave malignancy.

November 25, 1932. Due to suspicion of recurrence the patient was readmitted to hospital for biopsy of ear tissue. Specimen consists of three pieces of tissue taken from the ear. Sizes vary. Section made from ear. These sections show that the pieces consist almost entirely of tumor underlying skin, the tumor consists of fibroblasts with a few areas of myxoma. Also a few mitoses are seen.

Diagnosis: Myxofibro sarcoma of the ear.

We had been called in to see the patient in consultation and had taken the above biopsy for our own information. In view of the past history and the microscopic findings we felt that the most radical removal of the tumor was indicated with a complete removal of all ear cartilage, as we felt this to be the offending factor and so on. December 5, 1932, the patient was readmitted to the hospital for recurrence of tumor of the right ear.

Operation: Wide and deep excision of all tissue about the right ear, complete removal of all ear cartilage and a thorough radical mastoid operation was performed. An impression of this large cavity was then taken with dental modeling compound and a split skin graft inserted under the impression. The impression wax was removed in 10 days with complete healing of the wound.

Specimen: Consists of an external ear. The pinna measures 4.5 x 3.5 cm. The lobe measures 2 x 2 cm. The lobe appears normal but wrinkled. The tissue appears to be composed of fibrous tissue and clots.

Microscopic Examination: Tissue shows a small amount of hyperplasia and squamous epithelium on one side. The main portion of the tissue is composed of fibroblasts, most of which are adult in type. Deeper down there is a small amount of myxomatous tissue mixed with younger fibroblasts.

Diagnosis: Myxofibro sarcoma of the ear.

The removal of a myxofibro sarcoma of the ear should be early, wide and complete. If the cartilage element is involved a radical excision of this should be done. O. T. Roberg, Jr.,[1] claims that the presence of myxomatous tissue seems to be associated with inhibition of the invasive destructive tendency of chondro-sarcoma, are radio resistant and do not respond to radium or x-ray except the rapidly growing tumor with undifferentiated chondroblastic cells, these only show a fair degree of roentgen susceptibility.

This case demonstrated again very strikingly the futility of inadequate surgical procedures when dealing with malignant growths.

490 Post Street.

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XCIII

A NEW APPROACH FOR NASAL IMPLANTS*

SAMUEL COHEN, M.D.

PHILADELPHIA

This paper is not presented as a thesis for the correction of all saddle-noses, therefore a discussion of saddle-nose correction will not be gone into except to state that until a short time ago saddle-nose correction meant the simple insertion of some substance of proper size, shape and consistency to fill the depression. Today, however, this simple procedure is in many cases insufficient. These patients expect not only that their depression be filled and the dorsum leveled off, but a result that is cosmetically in harmony with their other facial features. They most often need rearrangement of the main nasal structures, narrowing of the nose at the bridge or changing the shape of the tip.

I will discuss only the technique used in those cases that require only the simple insertion of a transplant.

There are two main approaches for implants, the extranasal route, and the intranasal route. The main objection to the intranasal route is that postoperative infection is said to be more frequent. My experience does not prove this correct; if an intranasal entrance is made where it should be, within the skin lining of the vestibule, infection from skin incisions are the same, whether performed intranasally or extranasally. We must remember that as both are skin surfaces vestibular infections are treated similarly to extranasal skin infections.

The nasal vestibule is lined by skin of almost the same thickness as that of the skin of the nose. It can be prepared for operation just as well as the skin of the external part of the nose or areas nearby, by the use of iodine and alcohol or Tr. metaphen, which are not used on mucous membranes.

The nasal secretions can be prevented from coming into the nasal vestibule by proper postoperative packing, proper coadaption

*Presented before the Philadelphia Laryngological Society, October, 1937.

of incision lines, and no nose blowing for about one week post-operatively. The contact of the secretions with the entrance incision is avoided for a long enough period to permit the incision to become closed by reparative processes, thus preventing the entrance of infective organisms. So, I believe from the standpoint of possible infection, results will be the same whether one uses a vestibular incision or an extranasal incision.

Another claim against the intranasal route is that the tunnel, made from this entrance field, cannot be as straight as the external route. With care I have found it can be made as straight (of course the external route is much simpler from that point). I have used the intranasal route for many years, and my implants have been inserted straight and have so remained. I have had only one infection. This patient developed ether pneumonia which necessitated the use of an oxygen tent so that the local field could not be carefully watched.

The most frequent external routes used for implantations are:

1. The columellar route. Here an incision is made crosswise, or lengthwise, just under the nasal tip and above where the columella blends with the under surface of the tip. From there with fine scissors and dissectors a tunnel is made along the nasal dorsum, even to the glabella, when necessary. From the initial incision another tunnel can be made back to the anterior nasal spine for an additional implant to help raise the tip. A proper sized, shaped, and if necessary bent piece of cartilage or other substance can be inserted (the longer strip is for the dorsum and the shorter one for the columella).

2. Another external route, not used much now, is across the nasal dorsum, within the nasal-glabellar groove. Here you can burrow a tunnel down to the nasal tip, but you can't burrow along the columella to the nasal spine. In addition this leaves a more exposed scar than the columellar route.

Another entrance route, now very seldom used, is an incision at the inner edge of one eyebrow, and from there elevating the skin structures down to the tip. This has the same objections as the second external route mentioned and, in addition, there is a greater possibility of the tunnel not being straight, though the scar here is well hidden by the regrowth of hair of the eyebrows later on.

The external incision along the side of the bony dorsum, as far as I know, has been abandoned altogether.

These same entrance incisions have also been used for other operative procedures besides preparing a tunnel and bed for an implant.

The intranasal routes are mainly:

1. An antero-posterior incision along the lower border of the upper lateral cartilage within the skin lining of the nose, a la Joseph. This permits of a tunnel and bed being made to the glabella and by careful dissection it can be extended far into the nasal tip; extensions backward along the columella are of course not possible.

2. An incision in the nasal vestibule under the tip, lengthwise, near the lower anterior edge of the quadrilateral cartilage, and paralleling this anterior edge. From here a tunnel can be made to the glabella; but again not backward. This closes more quickly than the first, so that the implant becomes fixed in its new position more quickly. By being less exposed than the incision at the lower border of the upper lateral it is subject to less irritation. This entrance incision I have used for years with good results. The entrance incision that I will describe here has advantages over this last and I believe gives still better results.

Some of the objections to the old intranasal route are:

1. The question of infection from within the nose is to be considered.

2. The tunnel is too high above the tip so that the implant is often placed too high, and not only is it somewhat difficult to keep straight, but does not support the tip. Joseph used his Trelat knife to push forward from this tunnel to the tip. The tip skin is very adherent so that this elevation is difficult.

3. When an additional implant for the columella is needed to push the tip forward, an additional external incision has to be made.

4. The chance of the implant slipping is possible, but is less in my new approach than in the Joseph approach.

Through my incision a hinged cartilage can be introduced, so that one part becomes the new raised nasal dorsum and the other the reinforced columella.

A few statements on the anatomy involved are important for a better comprehension of the procedure to be described. The dorsum of the nose, that area from the glabella to the tip, is formed from above downward by the nasal process of the frontal bone,

the nasal bones and the anterior portions of the upper lateral cartilages, which are supported by the cartilage of the nasal septum. Below this, i.e., between the lower ends of the upper laterals and the nasal tip, is a small area where the dorsum is made up by the lower subcutaneous anterior edge of the septal cartilage. The tip of the nose consists of the greater or lower cartilages. These are made up of two plates; a lateral plate which forms the side of the nasal tip, and a twisted backward extension of this, the medial plate, which partly hugs the lower border of the septal cartilage near the tip, forming part of the columella. This is present on each side at the tip. All this bony and cartilaginous framework is covered by skin, subcutaneous tissue and some few muscle fibers. This superficial covering is rather loose from the glabella to the tip, very adherent at the tip and also quite adherent over the columella, but not as much as at the tip itself. The lower border of the medial plates is easily seen in each nasal vestibule and becomes prominent, when the side of the nostril is lifted up with a fine hand retractor or even by the finger. This lower border is about one-sixteenth inch to one-eighth inch away from the outer free margin of the nasal vestibule. It is at this lower border, near the tip, where the lateral plate makes its bend to form the medial plate, that is the area for the procedure to be described. Here no scar should be visible later on and ample room is provided for the necessary procedure.

Here an implant can be introduced and inserted along the nasal dorsum, and if necessary a pocket can be made to extend backward to the anterior nasal spine behind and in between both medial plates of the lower lateral for another implant, so as to push the nasal tip forward as much as is desired. I wish to stress the point that a columellar implant to be effective and give good results should be not between the two medial plates, but somewhat above them as near as possible to the lower border of the septal cartilage. Normally it is the septal cartilage that is the main support of the lower part of the nose and the tip, so that the columellar implant should be made to act like an extension of the septal cartilage and placed at the same level; then the end result will be more pleasing.

PROCEDURE

1. An incision is made within the nasal vestibule, far forward near the tip. The incision follows the outline of the lower border of the outer plate of the lower lateral as it is made to stand out in relief by the following method: The thumb of one hand lifts up

the outer margin of the nasal vestibule, while another finger of the same hand pushes the vestibular structures behind this into the vestibule. Another method of outlining the lower border of the lower lateral is to insert a small double-pronged retractor just under the edge and pull forward and upward on the outer vestibular margin, and pressure from without with the little finger immediately above the retractor, everts the lower edge of the lateral cartilage.

2. After the first incision has exposed the cartilaginous border well, a separation of this cartilage from the overlying skin is continued by means of a pair of fine curved scissors (by an opening and closing motion, very little actual cutting being necessary). The separation is extended in various directions so that a fair-sized pocket is produced under the superficial structures covering the tip. The opposite lateral plate can then be felt, but not seen, and the pocket may be extended above the opposite lower lateral.

3. With scissors or dissectors, sharp or dull, a pathway can be made upward, toward the frontal bone; as far as is necessary.

4. If a columellar implant is needed, a pathway can now be made from this original pocket backward until the anterior nasal spine is reached. This is best done, I believe, by a pair of fine scissors. The pathways are now probed, all clots are pushed out, and bleeding stopped, by external and internal pressure, for several moments. The tunnel or tunnels are then ready to receive the implants.

5. The implant is now placed in the position desired. The original vestibular incision can be closed by a fine silk suture, although this is not necessary. A small iodoform pack is inserted in the operated nasal vestibule, and an external nasal splint, fashioned of dental compound is placed over the nose (with an oiled gauze pad between the nasal skin and the mold). This dental mold is held in place by strips of adhesive plaster. The mold and iodoform plug are removed in two days. Healing is usually uneventful.

In conclusion it is noted that this approach also permits of various operative procedures upon the nasal framework, such as removing small bony or cartilaginous excrescences or other irregularities of a minor character, especially when the initial incision is repeated on the opposite side. If the incisions are made large, an entire nasal plastic can be performed as almost the entire nasal

covering can be lifted up like a curtain and work permitted underneath. This method of undermining the skin over the nose should be found useful in the correction of fairly large scars. The scar is removed and because of the wide undermining a minimum amount of tension will result when the new edges are approximated. Of course in small scars simple removal, slight undermining of edges, and good approximation will give good results.

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Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, March 7, 1938

THE PRESIDENT, DR. WALTER H. THEOBALD, IN THE CHAIR

The Biplane Fluoroscope in Bronchoscopy

PAUL H. HOLINGER, M.D.

(Author's Abstract)

Fluoroscopic aid is indispensable in directing certain types of endoscopic procedures. Grier of Pittsburgh, working with Jackson and Patterson, developed the first biplane fluoroscope used in conjunction with a bronchoscopic procedure, in 1907. Since that time such roentgenologists as Manges, Pancost, Pendergrass, and Chamberlain have made significant advances in this field.

The most common indication for fluoroscopically guided bronchoscopy is that of aiding in the removal of foreign bodies. The fluoroscope pictures the operative field more completely than it can be visualized through the tube and, therefore, the bronchoscope and forceps can be guided to the foreign body and the proper grasp secured. Small objects in the periphery of the lung, lying in the costophrenic angle, are lodged in bronchi so small that an ocularly guided bronchoscope will not reach them. Therefore, specially constructed 2 and 3 mm. costophrenic bronchoscopes and forceps must be directed to the foreign body by means of fluoroscopic aid. Other bronchial foreign bodies requiring fluoroscopic assistance are those which become embedded in the bronchial wall, those which have multiple points, or those lodged in the upper lobe bronchi. Nails, screws and tacks that are head up present a special problem because of the manner in which the mucosa encircles the head to make application of forceps difficult or impossible without fluoroscopic help.

The removal of foreign bodies from the esophagus, if they are opaque to the x-ray, is likewise facilitated by fluoroscopic guidance; open safety pins, dental prostheses, etc., are best handled in this manner. Finally, the endoscopic removal of foreign bodies from the

stomach can be done only with the aid of the biplane fluoroscope. Because of the relative safety of this procedure, gastrostomy is rarely if ever necessary for the removal of foreign bodies.

While foreign bodies are the most common indication for fluoroscopically guided bronchoscopy, the removal of tissue from tumors and the drainage of abscessed areas in the lung are facilitated in those cases in which the pathology is in an otherwise inaccessible location. In certain cases bronchography may be done under the fluoroscope, the opaque oil being more accurately directed into the diseased areas. Similarly, in perforating a stenosis of the esophagus, the procedure is made infinitely safer if both the upper tube, passed through the mouth, and the lower tube, passed through the gastrostomy opening, are visualized in two planes before the stenosis is punctured.

Contraindications for this type of work are the same as those that exist for any endoscopic procedure. However, it is the greatest fallacy to substitute fluoroscopic guidance for proper training with the open tube because, unless the operator is thoroughly trained in open tube work, he does not appreciate the dangers involved. The relative theoretical ease with which this procedure can be done makes it appear simple while, on the contrary, it requires the most elaborate type of team work and instrumentarium if it is to be done successfully and without danger to the patient or the operating team.

In order that the apparatus be of greatest efficiency there must be an accuracy of detail without distortion on the fluoroscopic screen, in two planes at right angles to each other. The apparatus must be shock proof and of absolute safety of operation, both to the patient and to the operator and his assistants, and must be flexible enough so that the center of each beam can be moved easily to visualize any part of the chest or abdomen. Framework about the table must be eliminated, as it obstructs the operator, the head holder, and the shoulder holder.

To meet these requirements, biplane fluoroscopes have been built for Research and Educational Hospital and St. Luke's Hospital on the principle of the fluoroscope built by Dr. Edward Chamberlain for the Jackson Clinic at Temple University Hospital. The apparatus consists of two Phillips water-cooled tubes, one for the vertical ray and one for the horizontal ray. Each tube is mounted four feet from the patient on a large framework support upon which the screen is also mounted. The support runs under the floor as a cradle for the vertical ray tube, rises perpendicularly behind a wall, three feet from the operating table, to support the horizontal ray tube,

then extends forward to hold the screen. This entire supporting framework is counterbalanced and mounted on wheels, and can be moved along the patient to keep the center of the beam on the center of the fluoroscopic screen. Thus the rays of each tube as they pass through the patient to the screen are parallel, there is a minimum skin irradiation, and all encumbrances around the patient are eliminated. The shutters are heavy enough to eliminate all irradiation except that small field which emits through the opening. The shutters are opened and closed by electric motors which are controlled from the screen. A second control regulates the lateral position of the vertical ray tube. Time clocks attached to each tube give the elapsed time as each tube is operated. Thus, the roentgen units of exposure to the skin can be accurately determined. The milliamperage can likewise be controlled directly from the fluoroscopic screen, permitting variations of from 2 to 8 milliamperes, depending upon the size of the patient and the location of the foreign body. A booster control permits more intense illumination when greater fluoroscopic visualization is required.

In order to take roentgenograms, a switch instantly changes the setting from the fluoroscopic to 115 KV and 100 milliamperes for plates. The timer is connected in series, and photographs can be made with a twentieth of a second exposure.

In order that greatest accuracy can be obtained, it is possible to turn the fluoroscopic table end to end, making the portion in which the foreign body lies closest to the screen.

This apparatus has been entirely satisfactory in every respect.

Representative foreign body cases were presented to illustrate the principles of biplane fluoroscopic guidance.

DISCUSSION

DR. A. H. ANDREWS, JR.: The speaker has been becomingly modest in not mentioning his own work in designing these fluoroscopes. Another point that might be emphasized is the painstaking work which is required in the localization of these foreign bodies. These cases run into minutes, sometimes an hour, and the infinite number of small bronchi which have to be explored makes it a very tedious and painstaking procedure and, unfortunately, repetition is sometimes necessary.

It is important to know the previous x-ray exposure in order to prevent overdosage. The biplane endoscopy may have to be shortened or postponed accordingly.

Seven-year Survey of Malignancies of the Head and Neck

NOAH D. FABRICANT, M. D.

(Author's Abstract)

A statistical survey of the incidence of malignant tumors about the head and neck as seen at the University of Illinois College of Medicine during a seven-year period (1930 to 1937) in which 249 cases were studied, establishes the following:

1. Malignant tumors occur seven times more frequently in males than they do in females.

2. The importance of bronchoscopy and esophagoscopy in enhancing and illuminating malignant tumor statistics.

3. Extreme youth and extreme old age, in either sex, show approximately the same malignant tumor involvement.

4. Most malignant tumors occur during the fifth decade of life, with the sixth and the four decades following in order.

5. The vast majority of tumors are of the squamous cell type.

DISCUSSION

DR. SAMUEL SALINGER: The thing that struck me as being unusual about this report is the high percentage of squamous cell carcinoma, particularly in the pharynx and nasopharynx. Other series of statistics that I have read, as well as my own observations, seem to show more of the epidermoid or the anaplastic cell variety. I, too, like Dr. Galloway, expected to hear something about the nature of the treatment these patients had undergone, and the results obtained. We are all pretty familiar with the etiology and distribution of these malignancies, and have been struggling to determine the best means of dealing with them. Naturally we want to learn all we can from the experiences of others. Two hundred and forty-nine is a pretty large number of cases and I am sure it would be interesting to know how they were treated and what results were obtained. I hope the author will make this the subject of a future report.

DR. FRANCIS LEDERER: I do not think the original purpose of this statistical study was to take up the question of treatment. We should really return the question to Dr. Galloway and to Dr. Salinger as to what their figures of cures reveal—that would be fair enough. In no uncertain terms, they are trying to put us on the spot.

A few points in this direction might not be amiss. Of the 57 laryngeal cases reported in this series, less than one per cent were oper-

able cases. After all, the type of malignant disease that comes to any clinic is in direct proportion to the lack of intellect and the size of the purse. Usually it is not the type that lends itself to operative intervention. If you are asking for radium and x-ray statistics, I have this to say. We have followed the results over a period of 13 years and I present our experiences very briefly to you. I do not want to discredit radium and x-ray because I have every confidence that when more is known about them relative to dosage and technique, we can expect improved statistics. We have followed Coutard technique diligently and yet a "cure" is still to be realized. Most of these cases have come to our clinic with recurrences even after a ten-year period. I do believe that we will have to evaluate our statistics on a more definite basis. We have had cases that came to the clinic after having been reported in the literature as "cured." Postmortems have borne out the recurrence of the malignancy.

The original intent of Dr. Fabricant's paper was merely to evaluate general statistical information. The results of treatment would not alone be difficult to evaluate, but I doubt their value any more than any other clinical survey where cases come so late and follow-ups are so poor. I, too, am amazed at the number of squamous cell carcinomas that were found. Sarcoma of the pharynx in most instances is lymphoepithelioma, which we used to term small round cell sarcoma. So far as nasopharyngeal tumors are concerned, you are right, most of them are of that anaplastic variety, but Dr. Fabricant did not divide his statistics into epipharyngeal, nasopharyngeal and hypopharyngeal sites. But we are startled by the incidence of malignancy. Of course, the apparent increase would have to be qualified from many angles. The university is a slough box for that sort of far-advanced material, so we will have to qualify some of those studies and not judge them too harshly, because we have to make allowances for many factors which alter statistical data. The present survey only includes the cases on which biopsy was performed.

I hope you will forgive the attempt at this sort of survey, but I think in the light of variations with statistics from other institutions, this series adds interesting data.

DR. MAX KULVIN: I would like to ask the doctor whether his statistics indicate where, in the female, the lesions were found to be most common.

DR. NOAH D. FABRICANT (closing): There are several omissions to a really thorough statistical study. I could check back and

answer these questions, but I do not have my original material. There was one angle I started to pursue, but ran up a blind alley: that was, how long these people lived. Our social service department is inadequate, or the people have moved or died, and I was not able to determine how long these people lived after they had undergone treatment in our institution.

The Development of the Paranasal Sinuses

A. A. ZIMMERMANN (by invitation)

(Author's Summary)

A review of the development of the olfactory organ and of the sinuses was presented by means of lantern slides, illustrating the most important steps of the process. It was emphasized that the primordia (anlagen) of the sinuses arise rather late during the prenatal period of development and that the origin of the frontal sinus, in particular, usually is delayed until postnatal life. The presentation was based mainly on recent literature, although some original material was included.

The embryonic period proper, which lasts to the end of the second month, is occupied by the differentiation of the main features of the nasal cavities. The origin of the primary and secondary nasal cavities was described. It was pointed out that the secondary or definite nasal cavities are formed by incorporation of the upper portion of the primary oral cavity through the establishment of the secondary palate. In relation to these processes, the change in position of the primary and secondary choanae was described. At the end of the embryonic period the lateral wall of the nasal cavities presents three main conchae (turbinates), but the primordia of the sinuses are not yet established.

The paranasal sinuses arise as localized epithelial sprouts or recesses of the nasal mucosa during the fetal period of prenatal development. This is primarily a period of growth of previously differentiated primordia of organs. Minor details of differentiation, however, also occur during fetal life. The establishment of the sinus-anlagen is an example of such delayed differentiation. Thus, the mucosal recesses leading to the establishment of the maxillary and sphenoid sinuses arise during the third prenatal (fetal) month. The epithelial sprouts for the ethmoidal cells are established during the fifth and sixth fetal months. The site of origin of the epithelial recesses becomes the ostia of the respective sinuses. Until birth the simple evaginations from mucosal recesses remain small and should

not be called "sinus" until they pneumatize the bones, after which they are named. Pneumatization of the respective bones is essentially a postnatal process.

The site of origin of the epithelial primordia of the various sinuses is as follows: The sphenoid sinuses arise as paired mucosal evaginations at the upper posterior portion of the nasal cavities. They extend posteriorly downward. They remain very small throughout the remainder of the prenatal period. Even at birth the mucosal diverticuli are in no relation to the cartilage or bony sphenoid. During the first few years of postnatal life the mucosal sprouts of the future sphenoid sinuses become surrounded by the posterior portion of the cartilaginous nasal capsule. The latter portion may be ossified and corresponds to Bertini's ossicles (Van Gilse). In the fourth year begins the resorption of the posterior nasal cartilage. Anterior lower remnants of the latter may persist at eight to ten years and represent the "conchae Bertini." In middle childhood the epithelial anlagen of the sphenoid sinuses expand into the sphenoid bone. Pneumatization of the latter occurs relatively fast after seven years. The final form if not the extent of the sphenoid sinuses is attained between 12 and 15 years.

The maxillary sinus arises as a lateral epithelial sprout from the hiatus semilunaris (infundibulum) of the middle meatus. The middle meatus is the most productive area for the establishment of sinuses, inasmuch as it gives origin to the entire group of anterior ethmoidal cells as well. The mucosal recess which constitutes the anlage of the maxillary sinus attains relatively greater size than any other sinus primordia during fetal life. It develops into a tubular structure, elongated in the sagittal direction. It was shown that already during the third fetal month, i.e., shortly after its establishment, the mucosal recess of the maxillary sinus differentiates glandular sprouts from its walls. At birth, the maxillary sinus constitutes a fairly well-developed tubular sac, the lower margin of which lies slightly below the level of the upper border of the inferior meatus. Anteriorly it extends to the lacrimal duct. The expansion of the maxillary sinus occurs at a relatively slow rate during early and middle childhood. With the seventh year the expansion of the sinus and pneumatization of the maxilla progresses at a faster rate. The first dentition exerts no influence upon the direction of growth or upon the form of the maxillary sinus. The second dentition, particularly the germs of the permanent molars and canine teeth, however, contribute to the molding of the floor of the expanding maxillary sinus. In children, there is usually no accessory ostium.

The ethmoidal cells may be subdivided into three groups: the anterior, posterior and postreme ethmoidal cells, according to their origin from the middle, superior and supreme meatuses. The cells derived from the middle meatus usually lie anterior to those which originate from the superior meatuses. The group of anterior ethmoidal cells is further subdivided into infundibular, frontal and bullar cells.

The original arrangement of these cell groups usually becomes more or less disturbed by dislocations during growth (unequal expansion, secondary division of some of the cells) which process has been described as "the struggle of the ethmoidal cells." There exist great individual variations, even at birth, concerning the degree of development of the various ethmoidal cells.

The infundibular cells are usually the anterior-most ethmoidal cells, one of which may pneumatize the agger nasi after birth. Above them are three or four frontal cells which originate from the upper portion of the infundibulum, i.e., the frontal recess. The bullar cells arise between the ethmoidal bulla and the base of the middle concha from a secondary furrow which was described as "superior semilunar hiatus" by Gruenwald. One of the bullar cells pneumatizes the bulla itself which latter structure, as well as the uncinate process, may be considered as secondary conchae.

Two or three posterior ethmoidal cells arise from the superior meatus, and one or several postreme ethmoidal cells may arise from the supreme meatus. The occurrence of a supreme concha has been reported with a frequency of 88 per cent in fetuses of eight to ten months, 73 per cent in children of 9 years, 26 per cent in adults. Some authors consider this structure as a secondary concha derived from the superior concha.

At birth, all groups of ethmoidal cells are fairly well formed. They are rounded epithelial recesses, separated from each other by relatively wide interspaces and bony septa. The ethmoidal cells grow relatively fast during the early years. They approach each other and expand particularly during the second year, mainly upward. Their round form becomes more and more changed by mutual compression, laterally they are flattened by the lamina papyracea. In the seventh year the ethmoidal cells are much larger and begin to pneumatize all available space (frontal bone, conchae, sphenoid bone). Between 12 and 14 years the ethmoidal cells attain their final form.

The genetic history of the frontal sinus is complicated by the fact that pneumatization of the frontal bone may occur from any one of several sources. Before pneumatization occurs it cannot be

determined which will be the specific site of origin of the frontal sinus. At birth there is no frontal sinus. A frontal recess of the middle meatus (upper anterior portion of the infundibulum) however, exists in 95 per cent of cases. Three or four frontal cells (of the anterior ethmoidal group) arise from the frontal recess. At the end of the first year the pneumatization of the frontal bone begins either by expansion of the frontal recess itself, by growth of one of the frontal cells or, finally, by expansion of a bullar cell. There are even other possibilities for the establishment of the frontal sinus. This explains the great variability which exists in the location of the frontal ostium in the adult. During the first years, growth of the frontal sinus is slow. At the end of the fourth year it is only the size of a pea. Faster rate of growth and change in form begins with the seventh year. The definite size and form of the frontal sinus is probably established at 20 years, although it may slowly expand to old age.

Concerning growth changes in the nasal cavities as a whole, it was pointed out that at birth the proportion in height of the ethmoid and maxillary portions is 2:1 and that the pharyngeal openings of the auditory tubes in the newborn, therefore, lie relatively lower, usually in the plane of the palate. The rapid growth in height of the maxilla during the first six months leads to a relative shift in position of that opening, as well as to a proportion of 5:4 between the ethmoid and maxillary portions of the nasal wall at the end of six months of life.

DISCUSSION

DR. LOUIS Z. FISHMAN: Will Dr. Zimmermann explain the idea of the sphenoidal sinus being an old sinus?

DR. FRANCIS LEDERER. I might say to those who do not know Dr. Zimmermann that he is closely allied with us in our specialty. He worked in conjunction with McCready on the ear of the opossum. Their work was published in the Journal of Anatomy. You can rely on the facts that he has gathered and presented to us here. Some of us have heard it before, and we could listen repeatedly with interest to the embryology of the nose and accessory sinuses presented in such an illuminating manner.

DR. A. A. ZIMMERMANN (closing): In answer to Dr. Fishman, I should like to explain further the term "conchae Bertini." The term is apparently not in use in English references concerning the development of the sphenoid sinus. The Germans, and Karl Peter in Handb. d. Anatomie d. Kindes particularly have accepted the term.

The "conchae Bertini" are small bilateral cells located in the posterior superior portion of the nasal cavity applied against the sphenoid bone. They were first described by the French author Bertin.

In postnatal life, the mucosal recesses which constitute the primordia of the sphenoid sinuses, extend slowly toward the sphenoid bone. They first reach the posterior portion of the cartilaginous nasal capsule, part of which may be ossified as the so-called "ossicula Bertini" (according to Van Gilse). The resorption of this partly bony capsule begins in the fourth year. Between the age of eight and ten, only ventral portions or spurs of that capsule remain. These excavations into the posterior nasal capsule constitute the "conchae Bertini." Van Gilse considers them as "palaeo-sinus," in contradistinction to the "neo-sinus" which is formed by the subsequent invasion of the sphenoid bone proper. The term is probably not important. Personally, I have never noticed these conchae.

Study of Frontal Sinus Ostium

(Motion Picture Demonstration)

O. E. VAN ALYEA, M.D.

(Author's Abstract)

My subject, being mostly on anatomy, seems to come as an anticlimax to Dr. Zimmermann's talk. I shall attempt to vary mine somewhat by considering the clinical application of this anatomic study. The motion picture was made from specimens which we have at the University, and an effort was made to show the normal variations present in a large group of specimens, approximately 200. We may conclude that those are similar to the variations we would see in our offices in 200 cases. The variations apply to the frontal sinuses, the size, the shape, and contour, and to the intranasal connections of the frontal sinuses. I have prepared a slide which deals with the clinical application of irrigation of the sinuses through the frontal ostium:

TYPES OF FRONTAL SINUSITIS WHEREIN IRRIGATION IS MOST EFFECTIVE

1. *Acute:* After 48 hours, if temperature is normal. One irrigation may suffice.

2. *Acute:* Late stage—two to three weeks. With severe pain persisting: two to four irrigations required.

3. *Subacute:* Six to eight weeks: pain gone: discharge, cough and stuffy nose persisting. Septal resection, infraction of middle turbinate: six to eight irrigations.

4. *Chronic:* Early—three to eight months, developing from one acute attack: minor tissue changes. Symptoms and surgery same as subacute. Ten to 20 irrigations.

5. *Chronic:* Old—apparent tissue changes. Symptoms and surgery same as subacute. Also enlarge frontal ostium then, if no improvement on irrigations, external operation.

6. *Recurring:* An attack with each head cold. One irrigation may suffice. Surgery, between attacks.

7. *Vacuum:* X-ray negative. One irrigation sufficient. Surgery between attacks.

8. *Latent or Doubtful:* No local symptoms. One sinus slightly hazy on x-ray. Diagnostic lavage to eliminate as infection focus.

In the early acute case a patient occasionally does not want to continue with the use of palliative remedies. Consequently, after 48 hours, if the temperature is normal, irrigation is a safe procedure. One such treatment usually suffices. Frontal sinusitis will often clear up spontaneously between the fifth and tenth day if nothing is done: however, after two or three weeks if the headache still persists, the patient demands treatment. Then it requires two to four irrigations at two-day intervals. Next in the classification is the subacute type. These are prolongations of acute infections and are often seen about the sixth or eighth week. There is drainage, a cough and a stuffy nose. Headache with local pain and tenderness are not prominent symptoms. In these cases we usually find some nasal deformity which should be taken care of. Surgery, such as infraction of the middle turbinate or septal resection, can be done at this time or after the infection has been cleared up with irrigations.

The early chronic cases are next. They are usually seen about the fourth to sixth month. In these cases there are few permanent tissue changes present and, usually, the condition is merely a continuation of an acute frontal sinusitis. The symptoms are the same as in the subacute: discharge, stuffy nose, and perhaps a cough. These cases usually require 10 to 20 irrigations, but they are curable. There is the old chronic type with more or less permanent tissue changes: the symptoms are the same as in the subacute. Surgery is also similar with the added procedure of enlargement of the frontal ostium.

Then, if there is no improvement after irrigation, external operation is indicated.

The recurrent type is next. In these the patient develops an acute sinusitis with each head cold. The attack usually clears up with one irrigation, and surgery is indicated between attacks. There is the vacuum headache, with the symptoms of sinusitis. Characteristic of this condition is Ewing's sign—pain on pressure on the floor of the sinus. These cases are usually associated with a blocked middle meatus and the ostium is closed. Passing a probe to the ostium is usually sufficient.

There is a group of cases that might be called latent or doubtful. Cases are referred to you by an internist or orthopedist who is searching for a focus of infection. One sinus might be hazy on x-ray and it is important to determine whether or not it is infected. In these cases one must irrigate the sinus in order to find out.

DISCUSSION

DR. THOMAS GALLOWAY: Has Dr. Van Alyea seen any bad results after irrigation within 48 hours?

DR. MAX KULVIN: In view of the numerous variations in the entrance to the frontal sinus, do you x-ray every patient that you probe?

DR. O. E. VAN ALYEA (closing): I have not seen any bad results from irrigation of early cases. I know that throughout the country rhinologists are inclined to stay away from the frontal sinus for the first week, or entirely. Nevertheless, I think if you are in the habit of irrigating maxillary sinuses, you can apply similar rules to the frontal. I do not see how the procedure applies to one more than to the other.

The percentage of cases one is able to probe is high. I think it is easier to locate the frontal ostium than the maxillary ostium. That is because you do not have the uncinate process to climb over in order to find the frontal opening, and it is merely a question of getting into the middle meatus, which can be done by shrinking down the middle turbinate. The ostium is usually wide enough to admit the cannula, which is thin. The inflammation seems to involve the ostium not so much as the structures below, and those are what you must pass through. It is rather difficult to get an x-ray in all cases of sinusitis you want to treat. If the symptoms are present, transillumination is an important aid. If the attempt to reach the ostium fails, x-ray is indicated because there may be no sinus there.

Otogenic Aspects of Arachnoiditis

S. L. SHAPIRO, M.D.

(Author's Abstract)

Abnormal accumulations of cerebrospinal fluid have been discussed in the literature under different terms: the case histories, although showing certain variations, have certain points in common, one of these being the uniform involvement of the arachnoid membrane shown at operation or autopsy. The author discusses the anatomy of the meninges and the formation of the cerebrospinal fluid and points out the paramount importance of ear infections in causing this condition, by virtue of the close relationship of the temporal bone to the dura of the base of the skull. The general etiology and pathologic forms are discussed. From a clinical aspect there are three varieties:

(a) Those instances where the history and symptoms suggest a brain tumor.

(b) Cases in which a diagnosis of brain abscess appears probable.

(c) Patients in whom there is a relatively sudden rise of intracranial pressure with severe symptoms, but with no focal signs.

All these groups may possess an interest for the otologist. Two histories are given, the first one of a patient in whom a diagnosis of a cerebello-pontine angle tumor was made by the neurologic department. The vestibular findings by the ear department indicated a posterior fossa lesion, but not an acoustic neuroma. Operation disclosed only cystic arachnoiditis. The second case was that of a child who developed signs of increasing intracranial pressure following a middle ear infection with horizontal nystagmus. The neurosurgical department diagnosed a cerebellar abscess: the ear department diagnosed a temporal lobe abscess chiefly because the dural inflammation at the time of the mastoid operation had been confined to the middle fossa. At operation no abscess was found in the cerebellum, but puncture of the temporal lobe disclosed an internal hydrocephalus. Both patients recovered.

The problems of diagnosis and management are discussed.

DISCUSSION

DR. PAUL A. CAMPBELL: I would like to ask one question. Dr. Shapiro made the statement that the type of nystagmus led him to believe there was pressure on the brain stem. I would like to ask the type of nystagmus.

DR. HOWARD BALLENGER: What percentage of these cases are relieved by lumbar puncture? I am under the impression that the great majority of these individuals are relieved by this simple procedure. If so, it is a diagnostic point of great value.

DR. A. A. ZIMMERMANN: I would like to know how extensive the area of occlusion in the subarachnoid space might have been in order to lead to a condition of internal hydrocephalus. A localized obstruction of the subarachnoid space on one side or the other presumably would be no cause for blocking the flow of cerebrospinal fluid from the foramina of Luschka and Magendie to the Pacchionian bodies.

DR. SHERMAN L. SHAPIRO (closing): In answer to the first question, the spontaneous nystagmus was a purely horizontal type, of a sustained character, and continued for several weeks and was obviously of central origin. As there was no ataxic characteristics, such as those described by Dr. J. Gordon Wilson for the cerebellar type, I felt from its appearance that it was due to some disturbance in the central vestibular nuclei in the brain stem.

There are no doubt a good many mild cases of arachnoiditis which clear up spontaneously and are not reported in the literature. The severe forms will not get well unless the dura is opened through the mastoid or through some other area on the skull.

There are some points about some of these cases that are not quite clear. Hydrops of the ventricle or internal hydrocephalus associated with ear infection nearly always gets well with a simple tapping of the ventricle. This fact speaks against an actual closure of the foramina by a meningitis exudate. The generally accepted theory is that given by Boeninghaus many years ago, namely, that the dilatation of the ventricle causes a mechanical block by distorting the foramina. Even if we believe the fluid to be a simple secretion from the choroid plexus, these cases may still be classified as arachnoiditis since Zand and other students have indicated that the choroid plexus, like the arachnoid, is at least partly of mesodermal origin.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, April 4, 1938

THE PRESIDENT, DR. WALTER H. THEOBALD, IN THE CHAIR

Papilloma of the Tonsil: Report of Three Cases

IRA FRANK, M.D.

(Published in full in September, 1938, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, page 715)

DISCUSSION

DR. HOWARD BALLENGER: I am under the impression that papilloma of the tonsil is somewhat more common than one would assume from Dr. Frank's review of the literature. It is possible that some of these small isolated polypi-like growths that one sometimes sees on the tonsil are not true papillomata. I have had a few of that type which I have called papilloma, but have not always checked by a microscopic section. It is probable that most of these cases are not reported in the literature.

DR. FRANCIS LEDERER: Two questions arise in connection with this presentation: First, as to whether in Michael Reese Hospital they routinely sectioned all tonsils to find these three cases recorded by Dr. Frank and, second, whether they were serially sectioned in order to be certain that such a report could be interpreted with any degree of accuracy. In this instance we again find lack of evidence for a fundamental basis for argument, when referring to the literature. As Dr. Ballenger said, I am certain that many more such instances are observed by clinicians than are recorded in the literature. I believe that if you canvassed the men who have observed these benign tumors, particularly those of papillary type, it would be found that such lesions are more common than this report would indicate.

DR. IRA FRANK (closing): I cannot answer Dr. Lederer absolutely. Since there were gross lesions, I am quite sure that only those cases showing something abnormal were sectioned. I, too, thought the condition occurred much more frequently than it does until I looked up the literature.

A Case of Streptococcus Meningitis With Streptococcemia: Recovery

ALFRED LEWY, M.D.

(Author's Abstract)

R. L. H., aged 14, operated on May 5, 1937, for acute mastoiditis, subperiosteal abscess. Discharged June 4, 1937, with dry ear and wound healed.

Readmitted on July 29, 1937. History of headache of two weeks' duration: vertigo and vomiting, blurring of vision. In coma two days before admission.

Examination showed a slight discharge from the right ear, tenderness in mastoid scar, stupor, neck rigidity; Kernig, Brudzinski, Babinski positive; facial twitching; paresis of all right extraocular muscles and of external rectus left. Blood examination showed hemoglobin 70 per cent; erythrocytes 4,000,000+; leucocytes 21,000; the urine contained albumin, a few granular casts and a few red and white cells. The spinal fluid under pressure showed 710 cells per c. mm.: hemolytic streptococcus in smear and culture. Four successive cultures of the spinal fluid were positive, the fifth being sterile. Three successive blood cultures were positive for hemolytic streptococci, the fourth being negative.

Operation: Radical mastoid. The dura was uncovered in the middle and posterior fossae, exposing the lateral sinus. The cisterna pontis lateralis was drained with iodoform gauze. Sulfanilamide was administered, the initial dose being 60 grains with sodium bicarbonate and 60 grains daily by mouth with two 5 cc. ampoules Prontysil intramuscularly daily for four days. This was then discontinued for three days and Fowler's solution, 2 minims t.i.d. was substituted, then sulfanilamide was resumed. Transfusions of small doses of whole blood were given on the second, fourth and seventh days. The temperature was normal on the thirteenth day. At this time the optic discs were still blurred, elevated 1.5 diopter and showed some flame-shaped hemorrhages. These eventually disappeared. Thereafter recovery was uninterrupted.

Five months later the patient is still somewhat anemic, but has resumed all normal activities. The left knee jerk is absent, otherwise neurologic findings are normal. She is still under observation.

DR. GEORGE S. LIVINGSTON: I recall a previous report by Dr. Lewy of a cured case of otitic meningitis in which drainage of the

lateral pontile cistern was used, but sulfanilamide was not. Two such isolated reports might make one wonder whether the surgery was not more important than the drug, but our general experience, of course, points in the opposite direction. Dr. Lewy has asked me to discuss sulfanilamide therapy from the standpoint of dosage. In cases of meningitis, adequate dosage is probably the determining factor in recovery. From experimental evidence we learn that to protect mice against a streptococcic peritonitis, a sulfanilamide concentration of 10 to 15 mgm. per cent in the blood stream is required. If we use these figures as a guide, we can then determine an approximately adequate dose, which should then be adjusted to the requirements of the individual case. It has been found that it takes 48 hours to attain the desired concentration of sulfanilamide in the blood. If we begin with the usually recommended dosage of three-quarter grain to each pound of body weight in 24 hours, then in two days we should make a quantitative determination of sulfanilamide. This is done by the colorimetric test devised by Marshall and his co-workers. In cases of meningitis the cerebrospinal fluid as well as the blood should be tested. If the amount is low and the clinical condition has not improved, the dosage should be increased.

Sulfanilamide has been found to be excreted at the same rate that it is ingested, and under such circumstances it is sufficient to test the urine content of the blood. In seriously sick patients, however, with meningitis or septicemia, there is a considerable derangement of renal function, so that the urine test is unreliable.

I believe that only by frequent quantitative blood determinations can we safely and intelligently treat a potentially fatal infection with sulfanilamide. Not all infections can be cured with sulfanilamide, but the recovery rate will be higher if we use these means of adjusting the dosage to the peculiar requirement of the individual patient.

DR. THOMAS GALLOWAY: Since the first of last year the County Hospital group has had nine proven cases of streptococcus meningitis recover by the use of sulfanilamide and its derivatives, one without operation, which is something that was impossible previously, so we cannot deny the effectiveness of the drug. As Dr. Lewy showed, however, proper surgery of the infective focus is still essential. As to the colorimetric test for blood level of the drug, it is not difficult and is routine at the Evanston Hospital. We try to get only a level of 5 mgm. per cent.

The routine doses at Cook County Hospital are three-quarter grain per pound of body weight for about three days, then one-half

grain on succeeding days. Recently we have used sulfanilamide in .8 per cent in salt solution given intravenously. It is interesting to know the amount of the drug being used; 12,000 five-grain tablets were used at County Hospital last month in the treatment of streptococcic infections, and Dr. Fantus is proposing that it be limited to serious cases because of the cost and because of the possibility of serious results from its use.

Dr. Francis Lederer: There are two points that occurred to me in Dr. Lewy's case report. He ceased giving sulfanilamide at the time the patient became cyanotic. I hoped that Dr. Livingston would talk of that point. In the modern concept of sulfanilamide treatment, the therapy must be continued despite the cyanosis which will then subside. Another point I wish to bring up, viz., has that child entirely recovered? Somehow or other, judging from the history which Dr. Lewy presented, one would suspect that the child may have a latent brain abscess. With the inability to make a proper recovery, indicated by the pallor and failure to gain weight, there should be a strong suspicion of this possibility.

Dr. M. Reese Guttman: One of the drawbacks in the use of large amounts of sulfanilamide has been the danger of methemoglobinemia. Recently an antidote for this condition encountered in the use of sulfanilamide has been found in methylene blue. Small doses are hypodermically injected three times a day. This has been found very effective in preventing the occurrence of sulfhemoglobinemia. When it has appeared clinically it has disappeared in about 24 hours after the use of methylene blue.

Dr. Alfred Lewy: I am very much obliged for this discussion. The important thing is to bring out the management of cases by sulfanilamide. We have operated for streptococcic meningitis for many years without any notable success until we began giving sulfanilamide, and in addition to the two cases of Dr. Galloway, which recovered without operation, I have one case and another of which I have knowledge in which operation was refused, and the child recovered. I think we have to give the drug credit for recovery in most cases. That is why it is so important that we understand the drug, how to use it, when to discontinue it and what the dosage should be in the individual case.

The question of brain abscess in this case has been uppermost in my mind, and for that reason the child is still under observation. The child lives out of town and the family is very poor, and I strongly suspect that the rather slow recovery may be due to lack of proper

food. I have tried to make arrangements with the County nurse to see that she gets the sort of food she should have. I shall continue to watch her for evidence of residual brain complications.

Psychiatric Therapy in Dysphemia and Dysphonia

JAMES SONNETT GREENE, M.D.
Medical Director, National Hospital for Speech Disorders
NEW YORK CITY

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DISCUSSION

DR. ELMER L. KENYON: We are all grateful for this interesting and valuable presentation. I first met Dr. Greene almost exactly twenty-seven years ago, in Berlin. We came to America and we have met but a few times since. We have taken different courses. I have taken a path which has attempted to help to build up a national movement for handling speech problems. Dr. Greene has stuck closely to his institute. The field covered by the author tonight is so large that a complete discussion will not be attempted. I wish I might talk about the different types of cases mentioned. Others in the work have handled them all, have handled them effectively and often not in the way described by Dr. Greene. With respect to stuttering, or stammering—it is a matter of fashion whether you use the one term or the other—there are perhaps six or eight definite theories with respect to the etiology of this disorder: of which one is Dr. Greene's. I have no doubt that none of these theories are true in their entirety. Thus treatment is still on an empiric basis and, for the individual physician, is determined largely by his own conception of etiology.

Dr. Greene's marked emphasis upon what he designated the "stutter type" of individual, upon which type of personality he believes stuttering depends, is certainly open to question. Ninety per cent of cases begin before the sixth year of life. Why the disorder starts is not known or, at any rate, not agreed upon. If the patient continues to stutter he is in a serious situation. He cannot make good in expressing himself. His peculiar efforts at speech constitute a subject for ridicule. He fears to talk. Whatever there may be of a "stutter type" in the stutterer seems to me to be chiefly a development of stuttering itself. This is borne out by the fact that no one can foretell which particular child, or adult, shall take on stuttering;

and also by the fact that after recovery the former stutterer takes his place in life, and usually appears no different from the rest of us.

Dr. Greene's success in treatment, namely, 70 per cent of the cases, is very fine. I have never seen published statistics which have been above 25 per cent of successes. As to my own treatment of stuttering I approach the problem from the psychophysiologic standpoint, and, when I can obtain complete co-operation, I have no failures. This attainment of success in treatment by such widely different methods further indicates how doubtful is Dr. Greene's "stutter type" conception of etiology.

DR. C. T. SIMON: I certainly have enjoyed Dr. Greene's presentation of this material. I should like to raise a question with Dr. Greene and make a statement of my own. The question is, how many cases has he found that have started after the age of six years? As Dr. Kenyon mentioned, practically all our cases are reported to start before the age of six, and we can get the complete family history. Further, it seems many children exhibit stuttering symptoms but never become stutterers; that somewhere something happens and the boy or girl changes into a stutterer. I wonder whether you have found any information in your case histories concerning the greater frequency among boys than girls. Why do more boys than girls fix the early symptoms into a permanent stutter? We have no satisfactory answer to that.

I think that in dealing with stutterers we should get away from the idea that the stutterer is abnormal or deviates sharply from the normal population. From every observation we can make, the stutterer differs from the normal only in terms of his speech. Some of us are beginning to think that the emotional abnormality we find in the adult stutterer in all probability is the result of the speech defect, rather than the reverse.

DR. JOSEPH C. BECK: I do not wish you to break out into laughter because this may sound funny, but I am going to tell the truth regarding my acquaintance with this man. He is too modest; this institute in which he is carrying on is an absolutely charitable one. One might have the idea that he must have a tremendous practice, as indicated by case 26,965, etc., but in a large city, in a large charitable institution that is not difficult to understand.

I would like to speak about this condition from a public standpoint, economic standpoint, and charitable standpoint, because many of these unfortunate people are without a job and hopeless, and they go away after he treats them ready to take a position in society.

Maybe it is some hocus-pocus he uses, I do not know, but they go away better and some are cured.

I met Dr. Greene up in Toronto three years ago at an outing on a boat which the Medical Society gave for us one day. He stuck by me all day and told me all the details of his work—more terms than any dermatologic professor ever expounded. That night the American Board of Examiners had a meeting and I came in and sat down next to Mosher, who said to me: "What's the matter with you, you are stammering!"

What interested me in this lecture tonight was the reference to aphonia, hysteric aphonia. We all used to treat them and say we cured them, again and again. I recently had a very disturbing case, a patient from Danville with a beautiful cyst of the vocal cord. I told Guttman to take it off, and he did. He told the patient to be quiet for awhile and she has been quiet ever since. She has normal cords and they move all right. I suppose the trauma, getting the instrument into the throat, produced that state of affairs. She was very hoarse from the neoplasm, but now that it is gone she is aphonic. It is an interesting subject, and I want to express my appreciation of having Dr. Greene here tonight.

I would like to ask if he has ever used hypnosis in any of his cases. Mention is made by Europeans about hypnosis in treating these patients.

Dr. A. H. Andrews, Jr.: Dr. Greene has used a term tonight which I think deserves more emphasis, namely: "organism as a whole." This is quite important from a philosophic standpoint. We have seen the results of his work in curing this type of patient. The same holds true in many other fields. We have been held back for many years by the Aristotelian concept of dividing a subject into parts, studying their various components and trying to add them up again and get the whole. We should try rather to get a concept of the whole. This holds true in organic as well as functional diseases and in other parts of the body as well as the larynx.

Dr. Austin A. Hayden: I would like to ask what Dr. Greene does for speech correction or speech adjustment in deafness, or for deaf people.

Dr. J. S. Greene (closing): In reference to imitation as one of the causes of stuttering, we can definitely say that the percentage of those who commence to stutter as a result of imitation is quite large, especially where there is an older stutterer in the immediate family.

Referring to Dr. Beck's question as to whether surgical measures should be instituted in voice cases before vocal treatment is undertaken, this of course depends entirely upon the nature of the condition. In the case of the vocal cord tumor just mentioned surgical procedure is indicated and should be followed by vocal treatment. In answer to his question about the value of hypnotism I wish to say that hypnotic therapy was given up a long time ago. In this country at the present time it is used only occasionally and simply as an experimental measure. One of the principal reasons for its disuse is that stutterers are introverts and it has been found that it is much more difficult to hypnotize introverted, self-centered individuals than the so-called extroverts.

In regard to the ratio of male to female stutterers, we have found a very uneven sex distribution. About eight times as many boys as girls stutter. There are obvious reasons for this. First and foremost is the fact that early environmental stress is never so hard on girls as on boys. The element of social competition enters into the life of even the youngest boys much more decisively than into that of girls. Boys are injected into an incomparably more strenuous atmosphere of group games in which the prowess of much older boys sets the standard. In other words, the social impact is stronger in the male sex, and it necessarily follows that stuttering is more common among boys.

Another factor in the uneven sex distribution of stutterers is that girls inherently possess a finer nervous mechanism, a higher rhythmic sense, better co-ordination, and a higher progressive trend, and for that reason they are less likely to lose their standard of organization under new environmental conditions.

Some people seem to have been born more awkward than others. The stutterer is one of these people. He shows his lack of co-ordination not only in speech, but in his general activities. For instance, a mother will often tell us that her stuttering child is more clumsy than the other children in everything he does.

Given such a child with a neuropathic diathesis and living in a neurotic environment, it is easy to see how almost any strong environmental impact will precipitate a general disorganization. Since speech is our finest psychomotor function, it is only natural that a certain percentage of these children should show this general disorganization through speech disturbance. These people are temperamental, stutter-type individuals who have a relatively high potential for the spread of emotional tone.

How to integrate these disorganized individuals is the basic problem of treatment. Speech is secondary, because stutterers can all talk when they are alone or with children or animals. I am sure Dr. Kenyon will bear me out.

The first thing we tell new patients at our Medical-Social Clinic is that we are not interested in their speech. This seems incomprehensible to a person who for twenty or thirty years has centered all his thoughts on that very thing. It is personality growth and rehabilitation that we are interested in, although corrective speech work must be done to counteract individual negative speech habits. Muscular relaxation and co-ordination are important phases of our therapy. The aim is to inculcate a sense of rhythm and harmony and diminish tension. We even use fencing to promote standardization, gracefulness and accuracy of movement.

Of course, the ultimate aim of all phases of our therapy is to promote emotional control and to change the stutterer's warped personality. We have found that stutterers respond under the right type of guidance, and the therapeutic measures carried out at our Medical-Social Clinic have demonstrated that they have tremendous developmental potentialities.

I see these people grow. They come in fearful and timid, many of them unable to hold positions. We have doctors, lawyers, accountants and engineers among our patients, all with good educations, but unable to meet the outside world. We give them standardization and organization. We change their entire emotional pattern, so that they are able to meet life objectively and on an adult level. That is the answer to the stutterer's problem.

In reply to Dr. Hayden's question about therapeutics for the deaf, we do not carry on such work on an extensive scale. We usually refer such cases to the League for the Hard of Hearing.

Abstracts of Current Articles

NOSE

Influence of Sulfanilamide on Infected Sinuses of Rabbits: Chemical and Microscopic Studies.

McMahon, Bernard J. (St. Louis), Arch. Otolaryng., 28:222-233 (Aug.), 1938.

Sulfanilamide administered to six uninfected rabbits and to 12 whose sinuses had been injected with hemolytic streptococci was recovered in substantial amounts from the tissues of the nose and paranasal sinuses and from the blood of practically all rabbits.

The reaction to sulfanilamide in the tissues of the nose and the paranasal sinuses of these rabbits was not cytologic.

The beneficial effects derived from the use of sulfanilamide in such infections must be the result of its influence on the organism itself or on its toxins in permitting more active phagocytosis by the polymorphonuclear leucocytes and monocytes.

Sulfanilamide should be as effective in the treatment of streptococcic sinusitis as in the treatment of streptococcic infection elsewhere in the body.

The signs of sulfanilamide toxicity which occurred were hyperpnea, apathy, vertigo and spastic and flaccid paralysis.

TOBEY.

Pathogenesis of Sphenoidal Sinus Mucocele with Presentation of a New Rare Case (Sulla patogenesi del mucocele del seno sfenoidale con descrizione di un nuovo raro caso).

Fornari, G. B. (Catania), Arch. di Otol., Rino. and Laring., 49:315 (June), 1937.

The following, more probable, theories on the pathogenesis of mucocele of the nasal sinuses are presented and discussed: first, cystic changes of the sinus mucosa; second, obstruction of the sinus ostium by inflammatory processes; and third, cystic degeneration of an ethmoid cell which later develops and, depending on its location, invades either the frontal or sphenoidal sinus.

Fornari considers at length the applicability of such theories to the sphenoid sinus and concludes that the genesis of a sphenoidal mucocele depends primarily on some form of inflammation of the sinus mucosa proper, plus the existence of a stenosis of the sinus orifice. The latter condition may be the result of a congenital mal-

formation, obstruction from inflammation of the posterior ethmoid cells or lastly, from a trauma in this region producing scar tissue at the ostium.

The rarity of a sphenoidal mucocele is due to the larger dimension of the ostium as compared to that of the ethmoid and to its anatomical arrangement when contrasted with the length and tortuosity of the frontal sinus ostium. And also, the area in the vicinity of the sphenoid ostium is least attacked by common inflammatory processes.

Only eight cases of primary sphenoidal mucocele have appeared in the literature and the author herein reports the ninth.

SCIARRETTA.

Congenital Atresia of the Pyriform Orifice, Causing Respiratory Insufficiency. Its Surgical Correction (De l'atrésie congénitale de l'orifice piriforme, cause d'insuffisance respiratoire; sa correction chirurgicale).

Escat, E. (Toulouse), O. R. L. Internat., 26:65 (Feb.), 1938.

Escat points out that aspiration of the alæ nasi is not infrequently associated with a decided narrowing of the bony structure of the anterior orifice of the nose, even though its caliber is normal after the turbinates are reached. To get rid of this inward projection, Escat operates under local anesthesia through an incision from one canine across to the other, subperiosteal separation upward as for a double Denker operation, and removal of the bony lip with rongeurs, taking care to avoid entering the antrum and also widening the nostril too much. Packing both nostrils for 48 hours secures accurate coaptation; occasionally the soft tissues of the nostril will require incision below to give complete success. The subgingival incision is closed with catgut or linen.

FENTON.

The Relation Between the Abnormal Pneumatized Sphenoid Sinus and Optic Neuritis. (Sui rapporti tra abnorme pneumatizzazione dei seni sfenoidali e neurite ottica.)

Giacobbi, L. (Radiological Institute, Modena), Riv. Oto-Neuro-Oftal., 14:317 (May-June), 1937.

Three patients with optic neuritis in whom the rhinoscopic examination disclosed no pathological process are reported. The x-ray plates revealed all sinuses clear, but the sphenoids were extremely large. In spite of the absence of pathology the sphenoid sinuses were opened through the posterior ethmoid cells. In two cases the optic neuritis was relieved immediately and the vision returned to normal; the third also improved considerably, but the author con-

sidered the failure of complete resolution to be due to delayed surgical procedure.

Giacobbi is not able to explain the mechanism of how a large pneumatized sphenoid may influence the optic nerve. He recommends surgical interference when x-ray plates show an opaque or a clear but abnormally large sphenoid sinus.

The author calls attention to an article published by Segura, appearing in the Annales d'Oto-laryngologie, who reports similar findings with good postoperative results in cases of retrobulbar neuritis.

SCIARRETTA.

PHARYNX

Treatment of Carcinoma in the Pharynx and Larynx by Irradiation.

Hamblen-Thomas, C., Brit. Med. J., 4051:447-448 (Aug. 27), 1938.

The author first emphasizes in all cases of carcinoma of the pharynx and larynx: (1) Operation should be done where possible; (2) all patients undergoing irradiation treatment should be treated as in-patients; (3) radium has its particular uses, and so has x-ray treatment.

The suitability of a growth for irradiation depends on its histological character and its situation. If cells are young and undifferentiated and the growth is rapid and large it is better treated by irradiation, and at the same time the results of operation alone are so unsatisfactory. On the other hand, if the malignancy is of the more mature type with a good deal of fibrous tissue, or has been treated previously by irradiation or operation so that there is an increase in scar tissue, it has little radiation sensitivity and is better operated on.

He believes that carcinomata arising in certain sites are especially sensitive to irradiation as: (1) Tonsil, if the growth has not become adherent to the more solid structures; (2) soft palate; (3) intrinsic carcinoma of the larynx. Where there is firm fixation of the growth and involvement of muscle and cartilaginous or bony tissues irradiation alone will not be successful.

If glands are present the results of irradiation are not so good since the glands are more resistant than the primary growth.

Irradiation has its uses in combination with operation; it destroys the young and active cells of the growth and renders an

adherent growth more movable. Quoting Coutard, "Operation should be performed before the twenty-fifth day after irradiation—that is, before the reappearance of any new cells and any skin reaction."

He limits his irradiation to radium needles and radon seeds, using wide external irradiation previous to insertion of these. Radium needles, he feels, gives a more sustained dose than radon seeds and the fenestration method is the best for interstitial irradiation. Care is required in the spacing of the needles if they are to be completely effective.

Intrinsic carcinoma of larynx and other growths involving the pharynx are unsuitable for interstitial irradiation.

In conclusion, he states that x-rays are the best treatment for rapidly growing carcinomata, and they are the only treatment for extensive growths and serious gland involvements.

SMIT.

LARYNX

Use of the Hyoid Bone as a Graft in Laryngeal Stenosis.

Looper, Edw. A. (Baltimore), Arch. Otolaryng., 28:106-112 (July), 1938.

This operation is proposed as a method of utilizing the hyoid bone as a graft in the treatment of laryngeal stenosis in certain cases.

The principle depends on embedding the left end of the attached hyoid bone between the incised thyroid cartilage, to act as a wedge in enlarging contractures and deformities of the larynx and to permit a better airway. This firm bony graft acts as a splint to weakened and deformed cartilage. The ease with which the hyoid bone can be exposed, detached and rotated makes the procedure practical.

A living, attached and accessible graft, with the blood supply to its upper part undisturbed, has advantages over a foreign embedded graft, such as cartilage from a rib, an ear or some other part of the body.

The operation is an improvement for treatment in certain cases of laryngeal stenosis resulting from injury in adults. It is not proposed as a perfect and immediate cure-all for every patient with laryngeal obstruction and has not been tried on children.

TOBEY.

Cancer of the Larynx.

Orton, Henry Boylan (Newark), Arch. Otolaryng., 28:153-193 (Aug.), 1938.

Early recognition and diagnosis of cancer of the larynx makes possible cure by surgical measures—laryngofissure, laryngectomy or lateral transthyroid pharyngotomy.

Since there is recurrence in 50 per cent of cases of subglottic cancer, it is believed total laryngectomy is preferable to laryngofissure in its treatment.

Dr. Orton does not agree with those who say that laryngectomy is a mutilating operation.

In Dr. Orton's experience laryngectomized patients have not been despondent; they have been a happy lot, getting a great deal out of life.

TOBEY.

Psychiatric Therapy for Dysphonia.

Greene, James Sonnett (New York), Arch. Otolaryng., 28:213-222 (Aug.), 1938.

While treating these patients the therapeutist must realize that the person's abnormal psychologic reactions are based on a physiologic foundation, that his thoughts, feelings and actions are governed by the defects and limitations of his anatomic mechanism, that although his condition shows no evidence of organic lesion, still there exists a form of physical inferiority which influences the organism as a whole.

Therefore, a composite therapy which considers all these factors, including the development of a more integrated and mature personality, must be instituted in order to obtain desired results.

In a case of psychophonasthenia, for example, the laryngologist alone cannot administer adequate therapy. Since the basis of this disorder is on a deep level, psychiatric collaboration is most advisable. At the same time, a course of special training should be introduced to counteract vocal anomalies brought about through misuse of the larynx.

In the case of the patient with a falsetto voice, the laryngologist must have both psychiatric and musical leanings in order to bring about complete reorganization.

The same therapeutic idea applies to hysterical aphonia. Instead of using shock, which occasionally proves useful but more often detrimental, it is far better to employ a psychiatric approach in order to obtain more lasting results and thus avoid possible disaster.

In conclusion, the author emphasized the fact that it is necessary to use both local and general therapy. The presenting physical symptom—defense mechanism—must be considered as secondary and not as the principal object of treatment.

A careful anamnesis will generally uncover the underlying factors of these physical symptoms. Psychiatric measures should be directed toward bringing the patient's unconscious motives into consciousness. When this is accomplished, fundamental changes in the personality occur. The patient then accepts himself as he is, making the most of his physiologic foundation.

TOBEY.

Chronic Inclusion of a Foreign Body as the Cause of Bronchial Stenosis and of Serious Pulmonary Complications. (Inclusion chronique de corps étranger comme cause de sténose bronchique et de complications pulmonaires graves.)

Prof. Vlavacek and Dr. Masek. Bronch., Oesoph., et Gastro., 2:100 (April), 1938.

A 17-year-old boy developed pulmonary symptoms and a right-sided pleural empyema for which a rib resection was done with good surgical results. Cough, expectoration, low grade fever, and moderate cachexia persisted and no evidence of tuberculosis, lung abscess, or bronchiectasis was uncovered. Roentgenograms were negative except for evidence of thickened pleura. Bronchography with lipiodol revealed a definite stricture in the right inferior bronchus. Bronchoscopy, on the second attempt, permitted the discovery and removal of a metallic foreign body (collar button) from below the stricture. A recheck of the history revealed that the boy had aspirated this foreign body nine years before, had suffered a minimum of immediate symptoms, had not informed his parents and had forgotten about the entire episode. He had been apparently well during the entire nine years. Practically complete recovery quickly ensued.

KLAWANS.

Neurenoma of the Larynx. (Neurinoma della laringe.)

Malan, A. (Torino), Valsalva, 13:417 (September), 1937.

This case is reported because of its rarity. The author, in searching the literature, has been able to find only seven cases. The patient, a female aged 34, reported to the clinic in a very poor physical condition, having progressive dysphonia and nocturnal noisy breathing but no dyspnea.

Laryngoscopy revealed a round, smooth mass the size of a walnut covered by normal mucous membrane. It was located in the right laryngeal vestibule extending under the aryepiglottic fold,

filling the right pyriform sinus, thus pushing the epiglottis superiorly and to the left.

This tumor was removed through a laryngo-fissure under local anesthesia. The growth was easily shelled submucously with very little hemorrhage and the patient made an uneventful recovery, retaining a good voice.

The histological examination of the specimen revealed the typical structures of a neurenoma (perineurial fibroma).

After reviewing the surgical procedures employed in all cases reported, the author firmly believes that for complete removal and best results, as far as function of the organ is concerned, the laryngo-fissure method is to be preferred.

SCIARRETTA.

EAR

Craniocervical Immobilization in the Treatment of Lateral Sinus Thrombosis (La inmovilizacion craneo-cervical en el Tratamiento de la tromboflebitis del seno lateral).

Munyo, J. D., Apolo, E., and Castellanos, A. Iglesias (Montevideo), An. O. R. L., Uruguay, 7:1-5 (Jan.), 1937.

After a careful review of the operative management of sinus thrombosis, the authors cite one very severe case, in which the sinus was opened and curetted thoroughly without ligation. After four days, the child's condition grew suddenly worse, and they adopted the procedure proposed in 1935 by LeMée and Richier—immobilization of the head and neck by a plaster-bandage helmet. With the action of the neck muscles suspended, the temperature dropped in 24 hours, and the "helmet" was removed after 13 days. They feel that extensive systemic infection is favored by free movement of the head and neck in all cases of this character.

FENTON.

Improvement of Hearing in Cases of Otosclerosis: A New One-Stage Surgical Technique.

Lempert, Julius (New York), Arch. Otolaryng., 28:42-97 (July), 1938.

A new technique for the improvement of hearing of patients with otosclerosis is offered. It is a delicate, highly technical surgical procedure designed to create a fenestra in the bony capsule of the external semicircular canal and to provide a mechanical means of keeping this newly created fenestra permanently open. To obtain the desired results it is essential that every step in this technique be patiently and

skillfully executed in its minutest detail. To bring this operative procedure to a successful conclusion it is absolutely essential that every surgical step be performed in the exact order described. Each step in this technique is the foundation for the next step, and unless each step is concluded successfully, the next step and therefore the rest of the operation are doomed to failure. This surgical technique should not be attempted by any young otologist unless he is especially trained for surgical measures of this type. Any otologist with a keen knowledge and understanding of the minutest details in the anatomy of the temporal bone, resulting from a large and varied personal experience in operation on it, will master this technique without any difficulty after seeing it carried out.

The endaural, antauricular approach to the temporal bone is employed for the following reasons:

1. The tympanomeatal cutaneous membrane which I have described can be obtained only when the endaural, antauricular approach to the temporal bone is employed.

2. To avoid the risk of postoperative infection, in addition to applying the strictest rules of asepsis, it is necessary to limit the surgical attack to the tissues directly concerned and thus reach the objective with the least amount of sacrifice of tissue. This is best accomplished by employing the endaural, antauricular approach.

3. Better visibility and accessibility of the desired surgical field are obtained by this approach.

4. The employment of the endaural, antauricular approach is followed by a convalescence involving the least possible social and economic inconvenience to the patient.

5. Cosmetically, the endaural, antauricular approach results in a status as near as possible to that preceding the operation.

Dr. Lempert has operated on 23 patients with otosclerosis by the described technique. In 19 cases a good practical improvement in hearing was obtained and maintained. In four cases in which operation was performed in spite of poor existing bone conduction, no improvement in hearing was obtained.

This surgical technique was carried out and all the postoperative dressings made under the strictest rules of asepsis. In no case was there a postoperative infection of the tympanic cavity, the mastoid cavity or the labyrinth.

Of the 23 patients operated on, the newly created fistula in the external semicircular canal remained open in 22. In one case the fistula began to show signs of closure at the end of ten days and was completely closed at the end of three weeks. Lempert believes that in the 22 patients in whom the fistula remained open it will remain permanently open. His previous experience has shown that when regeneration of bone takes place in a newly created fistula, it does so immediately, and clinical evidence substantiating this fact may be observed after the first deep dressing. At the time of each dressing the response to the fistula test becomes less marked, until about four weeks postoperatively, when the fistula test gives a completely negative result. When regeneration of bone does not take place, the response to the fistula test continues to remain strongly positive, showing no signs of decreasing intensity.

He believes that it is reasonable to assume that a fistula which has not shown signs of beginning regeneration of bone after two months will remain permanently open.

TOBEY.

Otitic Meningitis Cured by Sulfanilamide: Report of Four Cases (Meningites post-otitiques traités par le para-amino-phenyl-sulfamide; quatre malades guéris).

Hubert, C. (Paris), O. R. L. Internat., 26:324 (June), 1938.

Hubert presented four cured cases of otitic meningitis to the O. R. L. Society of Paris last April, as follows:

Age 6. Sulfanilamide used 67 days, 118 grams by mouth; 1.92 gms. (in 0.85 per cent solution warmed in a water-bath to insure dissolving the drug) by intraspinal injection after withdrawal of a few cc. of spinal fluid, 7 to 10 cc. at a dose.

Age 11. Sulfanilamide used by mouth 17 days, 48.5 grams.

Age 34. Sulfanilamide used 32 days, 139 grams by mouth; 2.89 grams in 0.85 per cent solution by intraspinal injection.

Age 17. Sulfanilamide used by mouth 41 days, 100 grams; 1.84 grams in 0.85 per cent solution by intraspinal injection.

The first three cases were hemolytic streptococci; the last, a pneumococcus mucosus case. These case reports are admirably complete, and furnish daily details of spinal fluid and blood findings. As against the usual mortality rate of 90 to 95 per cent, Hubert's proven results, like those of American clinicians, are most encouraging.

FENTON.

Vertigo: Its Neurological, Otological, Circulatory, and Surgical Aspects.

Brain, W. Russell, Brit. Med. J., 4054:605-08 (Sept. 17), 1938.

This is a very brief and concise article on the various aspects of vertigo.

The word "vertigo" by its derivation implies a sensation of turning either of the body or of its surroundings. Vertigo, in order to describe any subjective accompaniment of disordered equilibrium, whether this be a sense of rotation or not is defined by Brain as "the consciousness of disordered orientation of the body in space."

In discussion of the psycho-physiological aspect of vertigo he states that for consciousness the orientation of the body in space is normally an orderly dynamic relation between the bodily schema and the schema of the external world. Vertigo is the state of consciousness which arises when this relation becomes disordered. This he illustrates by the vertigo produced in ocular paralysis.

From anatomical and physiological considerations his classification of vertigo is as follows:

(1) Psychogenic Vertigo—In which there is no rotation, but a feeling as if one is going to fall or is walking on air. This is present often in anxiety neurosis or as a conversion symptom in hysteria.

(2) Vertigo due to Cortical Disturbances—May arise in petit mal epilepsy, migraine, or in localized cortical lesions.

(3) Vertigo of Ocular Origin—This occurs in ocular paralysis and diplopia. Looking from a height may also produce this type of vertigo.

(4) Vertigo of Cerebellar Origin—He states that in spite of a massive lesion in the cerebellum, vertigo may be slight or absent, especially if the lesion is limited to the lateral lobe. However, involvement of the inferior vermis will produce vertigo.

(5) Vertigo Due to Brain-Stem Lesions—Vertigo may be caused by vascular or neoplastic lesions of the brain stem, but is most strikingly seen when disseminated sclerosis involves the pons.

(6) Vertigo Due to Lesions of the Eighth Nerve—As in acoustic neuroma and pressure upon the nerve by abnormal vessels.

(7) Aural Vertigo—Under this, only Ménière's syndrome is fully discussed. The various etiological factors are described particularly that of water retention. His treatment consists of removal of exogenous poisons, eradication of foci of infection and correc-

tion of water metabolism. In resistant and incapacitating cases, the choice lies between alcohol injection of the labyrinth or division of the vestibular fibers of the eighth nerve. SMIT.

Otogenous Non-Purulent Encephalitis.

Jerlang, Erik (Copenhagen). J. Laryng. and Otol., 53:283 (May), 1938.

The author reports a case which came under his care and which ended in complete recovery. He takes occasion to review the history of these affections as reported in the literature. He points out that the diagnosis of otogenous encephalitis without abscess formation is extremely difficult. In pronounced cases, the symptoms are exactly like those of cerebral abscess. The onset may be sudden or quite slow. Temperature is of particular interest. In most cases, it is extremely high for protracted periods, although cases are on record in which it remained normal. No cases with true bradycardia have been observed.

Concerning therapy he states that otogenous encephalitis has, in most cases, been treated as a cerebral abscess because an abscess could never be excluded with certainty. The surgeon should bear this condition in mind and should not incise the brain without restraint in many different directions on the assumption that there must be pus to evacuate. SMIT.

Sulphonamide in the Treatment of Acute Mastoiditis.

Horan, V. G., and French, S. Gay, Brit. Med. J., 4061:942-943 (Nov. 5), 1938.

All cases of acute suppurative otitis media were placed on sulphonamide (Crookes') as soon as they were admitted. In addition they received the accepted treatment of the acute ear. This included myringotomy.

During the year following the introduction of this routine, the ratio of acute mastoiditis and acute otitis media in their series was reduced from 22.7 to 4.5 per cent.

The authors believe in spite of the possible sources of error in the figures published, the incidence of acute mastoiditis in the Royal Naval Hospital, Chatham, has been notably reduced. Since the only difference in the accepted treatment of the acute ear has been the introduction of routine administration of sulphonamide on admission, they believe it is reasonable to suppose that the aforementioned drop in mastoiditis is due to the drug. Therefore, the sulphonamide group of drugs have a real place in the treatment of acute suppurative otitis media.

SMIT.

Preoperative Management of Acute Streptococcic Mastoiditis.

Atkinson, E. Miles (New York), Arch. Otolaryng., 28:10-20 (July), 1938.

An argument is put forward for delay in operation in cases of acute streptococcic mastoiditis until localization of infection has occurred. Preoperative management and the indications for operation are discussed. The results of immediate and of delayed operation as they have appeared in Dr. Atkinson's practice are presented in a table and show that when the expectant treatment is adopted there are fewer complications, a lower mortality and a shorter period of hospitalization.

TOBEY.

MISCELLANEOUS

Osteomyelitis of the Sphenoid and Petrous Pyramid Following Adenoidectomy in an Individual Affected by Acute Mastoiditis. (Osteomielite dello sfenoide e della rocca petrosa da adenotomia in individuo affetto da otomastoidite acuta.)

Bozzi, E. (Milan), Arch. di Otol., Rino. and Laring., 49:395-412 (August), 1937.

This interesting article is written in detail. Three photomicrographs selected from serial sections of the petrosal pyramid are presented. The current literature is discussed and American writers are quoted extensively.

The case reported is of a boy, seven years old, giving a history of repeated tonsillitis complicated by bilateral otitis media and difficult nasal respiration. Admitted to the hospital for adenoidectomy, two days after the surgical procedure he developed left acute otitis media with otorrhea. The patient improved for three days but, while the aural discharge decreased on the left, otalgia and profuse otorrhea appeared on the right side. Toxic symptoms developed with marked rise of temperature, but without any evidence of chill and meningeal reaction. Despite a mastoidectomy, the symptoms failed to improve. Four days later revision of the mastoid operation was deemed necessary. The petrosal was cleaned, but the patient died after two days. Suppurative and necrotic osteitis of the right ethmoid cells, sphenoid sinus and petrosal pyramid was found at autopsy (suppurative leptomeningitis).

The author considers this case to be the first reported in the literature as primary osteomyelitis of the sphenoid bone following adenoidectomy and in which the infection originated from the nasopharyngeal region. He brings out that the typical symptomology and absence of pathognomonic signs caused error in the diagnosis of the case. This example proves the possibility of a petrosal lesion

independent of auricular inflammation. Bozzi stresses the necessity to differentiate a clinical from an anato-pathological petrositis and apicitis, and proposes not to use the terms petrositis and osteomyelitis of the petrosa indiscriminately. Petrosal osteomyelitis should be reserved only for those forms which do not follow infections originated from the middle ear or antrum.

SCIARRETTA.

Clinical and Histological Peculiarities Observed in Cases of Scleroma. (Su di alcune particolarità cliniche e istologiche osservate in casi di scleroma.)

Simonetta, B. (Modena), Boll. delle Malatt. Orecch. Gola. Naso, 55:361 (October), 1937.

This disease had been restricted to the eastern and southern sections of Europe for a long time, but during the last two decades, endemic cases have been found in natives of the northern and western districts.

Contrary to the usual, typical, course of this disease as presented in textbooks and the literature, the histories of six scleroma cases reported by the author indicated that the initial and most distressing symptoms were caused by the growth located in the upper trachea, larynx and nasopharynx. Only one patient had a synechia on one side between the inferior turbinate and septum. Nasal symptoms always appeared as simple atrophy with copious discharge and crusting. Tumefaction and obstruction of the chambers were absent or appeared in the advanced stages of the malady.

These atypical signs have also been reported by other European observers. Due to the scarcity of cases, the author is unable to say whether this reverse symptomology can be attributed to the territory now invaded.

Simonetta briefly discusses the literature on this clinical point and extensively presents the histogenesis of the Mickulicz cells. He quotes the work of Streit and Schwedkowa-Rosche. He advances the opinion that only a few of the Mickulicz cells originate from epithelial cells. These have a polyhedral or oval shape with some residue of protoplasm at one or both poles of the cell. The majority derive from leucocytes which have migrated into the epithelial sheaths and possess the typical characteristics of the cells described by Mickulicz, i.e., a large, round, vacuolated cell with no trace of protoplasm and with or without fragments of a pyknotic nucleus at its periphery.

SCIARRETTA.

Tomography in the Field of Otolaryngology. (Tomografia nel campo dell'otorinolaringologia.)

Liveriero, E. (Torino), Valsalva, 13:305 (July), 1937.

The author describes the principles of tomography; also called stratigraphy or planigraphy. By this method only a special anatomical region, selected for demonstration, is clearly and distinctly separated from the other structures of the head.

Six roentgenograms accompanying the article illustrate results obtained with this technique. They present in detail the structures of the sphenoid, third ventricle, ethmoid cells, frontal and maxillary sinuses and the construction of the internal ear and mastoid cells.

A special mechanical equipment is necessary for taking these x-ray plates. Probably this procedure will be adopted extensively in the otolaryngologic field.

SCIARRETTA.

Alcohol Injection in Inoperable Malignant Growths of the Jaws and Tongue.

Harris, Wilfred, Brit. Med. J., 4059:831-832 (Oct. 22), 1938.

In the later stages of malignant growths, after treatment has been given and recurrence has appeared, it becomes necessary to make the remainder of the patient's life as bearable as possible. This can be accomplished in those cases in which the growth invades the territory of the fifth cranial nerve and where the growth has not spread into the neck or ear.

For the alleviation of pain in these regions, the Gasserian ganglion is injected either by the anterior or lateral routes with 90 per cent alcohol, approximately ten minims being used. The author believes that the anesthesia of a well-placed injection should last as long as the patient's expectation of life. His method is illustrated by seven cases of malignant growth of the maxilla and antrum, and one case of the tongue.

SMIT.

Books Received

Diseases of the Nose, Throat and Ear.

By W. Wallace Morrison, M.D., Clinical Professor and Chief of Clinic, Department of Otolaryngology, New York Polyclinic Medical School and Hospital. Cloth. 8vo. of 675 pages, with 334 illustrations. Philadelphia; W. B. Saunders Company, 1938. Price $5.50 net.

This is one of the most satisfactory texts which it has been our fortune to examine. Written for the undergraduate student and the general practitioner, the author has achieved a conciseness and simplicity of exposition and a completeness of material which make for easy consecutive reading as well as for really informative reference.

The discussions are strictly impersonal, as befits a textbook and the modern viewpoint has been presented clearly and briefly.

The value of any book, as a reference work, depends largely upon the detailed effectiveness of its index. A text whose contents are not quickly available soon finds a place on an upper shelf. Beside a general index of twenty-seven pages, there is a symptom index, itself an instructive part of the work.

Each chapter is followed by an adequate list of sources. This seems to us an extremely important inclusion in a textbook, for while the nature of the book forbids a detailed description of the researches upon which its statements are based, the student as yet unfamiliar with the literature should be assisted in looking into these original sources in the brief time at his disposal.

The first thing to catch the eye in Morrison's book is the treatment of the illustrations, all of which are line drawings. Closer scrutiny reveals them to be the work of the author. His "limited artistic ability" for which he apologizes in the preface has nevertheless succeeded in producing diagrams of great clarity and their homely simplicity adds something to their effectiveness as teaching material.

The book should enjoy wide popularity.

Notice

THE AMERICAN BOARD OF OTOLARYNGOLOGY

An examination was held in Washington, D. C., October 7th and 8th, 1938, prior to the meeting of the American Academy of Ophthalmology and Otolaryngology. One hundred and twenty-nine candidates were examined—of this number, ninety-seven were certificated.

During 1939 examinations will be held in St. Louis, May 12th and 13th, prior to the meeting of the A. M. A., and in Chicago, October 6th and 7th, preceding the meeting of the American Academy of Ophthalmology and Otolaryngology.

Prospective applicants for certificates should secure application blanks from the Secretary, Dr. W. P. Wherry, 1500 Medical Arts Building, Omaha, Nebraska.

H. P. MOSHER, M.D.
President

W. P. WHERRY, M.D.
Secretary-Treasurer